Utah State University
DigitalCommons@USU

All Graduate Theses and Dissertations | Graduate Studies

5-1981

Biofeedback: A Possible Substitute For Smoking

Earl Eugene Griffith
Utah State University

Follow this and additional works at: https://digitalcommons.usu.edu/etd

 Part of the Psychology Commons

Recommended Citation

Griffith, Earl Eugene, "Biofeedback: A Possible Substitute For Smoking" (1981). *All Graduate Theses and Dissertations*. 5897.
https://digitalcommons.usu.edu/etd/5897

This Dissertation is brought to you for free and open access by the Graduate Studies at DigitalCommons@USU. It has been accepted for inclusion in All Graduate Theses and Dissertations by an authorized administrator of DigitalCommons@USU. For more information, please contact digitalcommons@usu.edu.

BIOFEEDBACK: A POSSIBLE SUBSTITUTE FOR SMOKING

by

Earl E. Griffith

A dissertation submitted in partial fulfillment
of the requirements for the degree

of

DOCTOR OF PHILOSOPHY

in

Psychology

Approved:

UTAH STATE UNIVERSITY
Logan, Utah

1981

© 1981

EARL EUGENE GRIFFITH

All Rights Reserved

ACKNOWLEDGMENTS

I would like to thank Dr. Edward Crossman, Chairman of my Doctoral Committee, for his invaluable assistance, support, encouragement, and patience throughout this endeavor. I would also like to thank Dr. Sebastian Striefel, Dr. Glendon Casto, Dr. Michael Bertoch, Dr. Richard Powers and Dr. Glen Latham for their support and generous patience while serving on my dissertation committee.

I would like to extend special thanks to Dr. Sebastian Striefel and Dr. Marvin Fifield for allowing me to conduct this research at the Exceptional Child Center's Biofeedback Lab; and to Mark Schaefer, Ray Reitz, Fred Browning, and John Zukin for their assistance in the collecting of the data.

I would also like to thank the Utah Lung Association for the research grant they provided.

Last but not least I would like to extend a special thanks to my parents: Earl A. Griffith and Bettie Elizabeth Griffith for their support, patience, and trust in me throughout the completion of my entire academic career and this final important paper.

Earl Griffith

TABLE OF CONTENTS

TABLE OF CONTENTS (CONTINUED)

TABLE OF CONTENTS (CONTINUED)

LIST OF TABLES

LIST OF FIGURES

ABSTRACT

Biofeedback: A Possible
Substitute For Smoking

by

Earl Eugene Griffith, Doctor of Philosophy

Utah State University, 1981

Major Professor: Edward Crossman, Ph.D.
Department: Psychology

Numerous agencies have accumulated evidence since 1964 which implicates habitual cigarette smoking as a causal or facilitating factor in the development of many circulatory and respiratory diseases. This study sought to identify those psychological variables which possibly contribute to the maintenance of cigarette smoking and therefore, had two main purposes. First, this study investigated the individual and simultaneous physiological changes, i.e., Electroencephalography, Electromyography, Heart Rate, Blood Pressure and Skin Temperature that occurred during and immediately after the smoking of one cigarette. Second, the study investigated the hypothesis that smoking frequency would decrease when individuals were trained via biofeedback procedures to increase 8-12 Hz occipital EEG activity as a substitute for smoking.

Three male, very heavy smokers (35 or more cigarettes per day) and three male moderate smokers (15-24 cigarettes per day) physiologies were monitored while smoking, non-smoking and while they were provided with 8-12 Hz occipital EEG biofeedback training using a multiple baseline design. Results of the study indicate that of the six smokers physiologically monitored, four or more of the smokers demonstrated the following physiological changes while actually smoking one cigarette: the percent of time producing 4-8 cycles per second brain waves increased (S2,S3,S5); heart rate (beats per minute) increased (S1,S2,S3,S4,S5,S6); and the percent of time producing 8-12 cycles per second (Hz) brain waves decreased (S1,S2,S3,S4,S5,S6).

Immediately after the smoking of one cigarette, four or more of the smokers demonstrated an increase in their rates (S1,S2,S3,S4,S5, S6) and subjects 1,4,5 and 6 demonstrated an over-the-entire-session decrease in their skin temperatures. There did not appear to be any specific consistent brain wave changes across the subjects. However, the following subject-specific brain wave changes were evident: Subject 1 data indicates an increase in Alpha brain waves (8-12 Hz), a decrease in Theta brain waves (4-8 Hz), and a decrease in Beta brain waves (12-20 Hz). Subject 2 data indicates a decrease in Alpha brain waves, an increase in Theta brain waves, and a decrease in Beta waves. Subject 3 data indicates an Alpha wave decrease, Theta wave increase, and Beta wave increase. Subject 4 data indicates an Alpha wave decrease, Theta wave increase, and no observable change in Beta activity. Subject 5 data indicates an Alpha increase, a Theta decrease, and no observable change in Beta activity. Subject 6 data

indicates an Alpha decrease, a non-observable change in Theta production and an increase in Beta activity.

During the training period, when the smokers were given music feedback whenever they produced 8-12 Hz, four of the six smokers learned to increase the percent of time producing 8-12 Hz, (S1,S2,S5, S6). Two of these four smokers were able to continue producing high levels of 8-12 Hz activity without the use of biofeedback equipment (S1,S2). These smokers had quit smoking completely at the end of a six-month follow-up period. These two smokers were contacted by phone at the eight-month follow-up period and reported they were still absent from any cigarette smoking. The four smokers who could not increase their 8-12 Hz activity without the use of 8-12 Hz auditory feedback (Phase D) decreased their frequency of cigarette smoking at the six-month follow-up period as follows: Subject 3, from 38 to 15 cigarettes smoked per day; Subject 4, from 50 to 44 cigarettes smoker per day; Subject 5, from 18 to 8 cigarettes smoked per day; and Subject 6, from 17 to 10 cigarettes smoked per day.

Possible reasons why Subjects 1 and 2 quit smoking are discussed and directions for future research are presented.

(190 pages)

INTRODUCTION

Experimental and epidemiological evidence accumulated since 1964 by such agencies as the American Medical Association, the American Cancer Society, the American Heart Association, the National Tuberculosis and Respiratory Disease Association, the American College of Chest Physicians, the American Dental Association, and the American Public Health Association clearly implicates habitual cigarette smoking as a casual or facilitating factor in the development of lung and bladder cancer, coronary heart disease, cardiovascular diseases, emphysema and chronic bronchitis (U.S. Department of Health Services, 1971-1975). The health agencies have succeeded in making this information available to the public (Gallup, 1974). However, the effects of this information upon actual cigarette use have been minimal (Auger, Write & Simpson, 1972; O'Keefe, 1971). One of the apparent problems is that although some individuals have succeeded in discontinuing smoking, the majority of the smokers wishing to quit have been unsuccessful (Guilford, 1966).

Recent reviews of the psychological treatment of smoking (Bernstein, 1969; Hunt & Bespeloc, 1974; Hunt & Matarazzo, 1973; Kentzer, Lichtenstein & Mees, 1968; Lichtenstein & Kentzer, 1971; McFall & Hammer, 1971) indicate that numerous treatment techniques produce similar short-term (3 months or less) reductions in smoking rate, but that no apparent long-term reductions have been demonstrated (Hunt & Bespeloc, 1974; Hunt & Matarazzo, 1973).

In addition to the lack of treatment techniques demonstrating any long-term reductions in cigarette smoking frequency, there also appears to be some controversy among investigators as to the specific effects smoking has on the EEG of humans. One group of researchers (Brown, 1974; Itil, Ulett, Hsu, Klingenberg & Utlett, 1971; Phillips, 1971) suggest that smoking acts as a depressant and slows down brain wave activity, while another group of researchers (Murphree, Pfeifer, & Price, 1967; Ulett & Itil, 1969; Hauser, Schwartz, Roth & Bickford, 1958) suggest that smoking acts as a stimulant and speeds up the brain wave activity.

Biofeedback training, a new and promising technique for establishing voluntary control over many physiological processes, has been effective in demonstrating that by changing some physiological processes one can produce a change in some overt behavior, e.g., electromyographic (EMG) feedback training resulted in decreases in the frequency of tension headaches (Budzynski, 1973; Budzynski, Stoyva, Adler, & Mulloney, 1973); electroencephalographic (EEG) feedback training of the sensorimotor rhythm led to a decrease in the frequency of epileptic seizures (Lubar & Bahler, 1976). However, currently there are only three studies (Havelick, 1977; Kothare, 1975; Turin & Nideffer, 1974) which have tried to eliminate smoking behaviors through the use of biofeedback procedures.

Brown (1974) noted that individuals who have developed a habit of smoking cigarettes consistently demonstrate an increased frequency of 8-12 Hz activity as compared to non-smokers. In

conjunction with Brown's findings, the author's pilot research, which investigated the effects of smoking a cigarette on the physiologies of four moderate smokers, indicated that there was also an increase in the percent of time (8-12 Hz) brain waves were produced as a result of smoking a cigarette. Therefore, Brown's (1974) data and the author's pilot data suggest that 8-12 Hz activity might be a significant contributing factor in determining the frequency of cigarette smoking.

In summary, there is evidence which indicates a need for: 1) treatment techniques which produce long-term reductions in smoking rate; 2) additional research which investigates whether the smoking of a cigarette acts as a stimulant or depressant on the EEG of humans; and, 3) the investigation of the utility of biofeedback as a treatment technique for the reduction of smoking behavior.

The present study has two purposes. First, the design of the study will reveal the individual and simultaneous physiological changes, i.e., EEG (brain wave patterns), EMG (muscle tension), electrocardiogram (heart rate), electrosphygmomanometer (blood pressure), and skin temperature that occur during and immediately after the smoking of one cigarette. Second, the study will determine whether smoking frequency decreases when individuals are trained via biofeedback procedures to increase their amount of (8-12 Hz) occipital EEG activity. In essence, the study might provide smoking researchers with valuable information concerning the simultaneous physiological effects of smoking one cigarette. Data which will indicate the feasability of replacing cigarette smoking with a

voluntarily altered 8-12 Hz occipital EEG brain wave pattern will also be gathered.

REVIEW OF RELATED LITERATURE

The review of literature which follows shall not attempt to exhaustively review the theoretical or applied literature on smoking behavior, nor will it attempt to evaluate the effectiveness of each category of treatment techniques in depth, but instead is primarily designed to point out the major categories of controlled, experimental research relating to the modification of smoking behavior. As presented in the preceeding section, the major problem of the majority of the treatment techniques is their lack of long-term reductions in the frequency of cigarette smoking (Hunt & Bespeloc, 1974). Hunt and Matarazzo (1973) evaluated the effectiveness of the different treatment techniques, and presented some summary data on their relapse rates. Hunt (1974) notes that the average study presents results that indicate the percentage of successful abstainers decreases from 100% at the completion of treatment to less than 48% at three months, and only 25% or less of the smokers have actually quit smoking at the end of six months.

To date, the scientific world believes very strongly that cigarette smoking is a health hazard and that effective smoking therapies must be developed. In fact, the scientists believe it so strongly that the following statement appears on each pack of cigarettes: "Warning. The Surgeon General has determined that cigarette smoking is dangerous to your health." This single sentence

expresses a scientific judgment supported by a series of five Public Health Service reports issued since 1964. The first of these was the famous Surgeon General's report on Smoking and Health. This was the work of a committee of ten distinguished scientists appointed by the Surgeon General with the approval of President John F. Kennedy. Four (4) subsequent reports entitled The Health Consequences of Smoking, have now been issued (in 1967, 1968, 1969, and 1971). Together with the original Surgeon General's report, the following comments have been researched and documented.

1) The diseases most closely associated with cigarette smoking are lung cancer, coronary heart disease, emphysema, and chronic bronchitis. In other words, cigarette smoking affects mainly the respiratory and circulatory systems.

2) As might be expected, cigarette smokers have more disability and illness than non-smokers. They suffer more frequently from chronic conditions and spend more time sick than non-smokers. One estimate is that 77 million work days are lost each year in this country because smokers have higher rates of illness than those who do not smoke.

3) At every age from 35 years on, death rates are higher for cigarette smokers than for non-smokers. This is true of women, as well as men, and the differences are striking. Among men between 45 and 54 years, the death rate for smokers is almost three times that of non-smokers.

4) The more one smokes, the greater is the risk. Compared to the non-smoker, the two-pack-a-day smoker has more than twice

the change of dying of heart disease and 20 times the chance of dying of lung cancer than non-smokers. The effect of smoking is not, however, restricted to the heaviest smoker alone. The average smoker (one-pack-a-day), and the fairly light smoker (one-half-pack-a-day) can be significantly affected.

Different Approaches to the Modification of Smoking Behavior

Clinics

Since the early 1960's when Ejurp (1963, 1964) began his pioneering work, smoking cessation clinics have been a popular means through which smoking reduction has been attempted. Although there are many variations across clinics (e.g., treatment techniques, number and length of meetings, expense of treatment) most of the clinics involve groups of smokers coming together for the sole purpose of reducing their smoking behavior. The majority of the clinics attempt to achieve smoking reduction by: (1) presenting health information, (2) providing group therapy, (3) providing moral support, (4) providing social pressure, or (5) implementing any combination of these (Cruickshank, 1963; Hoffstaedt, 1964; Lawton, 1967; McFarland, 1965; Schwartz & Dubitzky, 1967).

Hypnosis

Hypnosis, as defined by Crasilneck and Holt (1976), is an altered state of consciousness which can be used in some individuals to produce desirable change in habit patterns, motivation, self-image and

life style. Johnston and Donoghue (1971) in their review of hypnosis and smoking, indicated that hypnotic techniques have been used as a part of antismoking interventions for the past thirty years, either to uncover personality conflicts which are presumed to cause smoking behavior, or to provide various kinds of direct suggestion (Bryon, 1964). Hypnotic suggestions have been used to give cigarettes an aversive taste or smell, to associate smoking with aversive events, to associate positive events with nonsmoking and, in general, to increase subjects' motivation for gradual smoking reduction, self-monitoring, stimulus control, response chain disruption, and a variety of other self-control tactics (Bernstein & McAlister, 1976).

Some reports of short-term success with hypnosis using combinations of the above approaches are: Kroger (1963), Perry and Mullur (1975), Von Dedenroth (1964a, 1964b, 1968), and Watkins, (1976).

Sensory Deprivation

Sensory deprivation techniques which have been used to modify smoking behavior usually involve attempting to reduce sensory stimulation to an absolute minimum, while utilizing a variety of persuasive communication techniques. Although the communication techniques vary, as to the methods used to reduce sensory stimulation, the communication techniques are usually related to persuading the client that the health hazards of smoking far outweigh the pleasures of smoking.

Sensory restriction procedures can be as uninvolved as having the subject lie quietly in a dark attenuated room with arms and hands encased in gloves and wearing earplugs (Schultz, 1965). Perhaps a more severe procedure to bring about total deprivation was that used by Lilly and Shurley (1961) and Shurley (1963), who immersed subjects in a pool of slowly circulating tepid water. The subjects were wearing nothing but a mask covering their eyes and ears, and were instructed to inhibit all movement (Schultz, 1965).

Whatever the technique used, the sensory deprivation approaches have notedly influenced the subjects' verbal behavior about smoking (Gallup, 1974; Janis & Mann, 1965; Leventhal, 1968; Lichtenstein, Kentzer & Himes, 1969; Mann & Janis, 1968; Platt, Krassen & Mausner, 1969; Streltzer & Koch, 1968). However, only a few studies using sensory deprivation techniques have actually shown a significant reduction in smoking behavior (Suedfelt, 1973; Suedfelt & Ikard, 1973).

Social Learning Approaches

Some reviews conclude that learning approaches to the modification of smoking behavior are the most promising (Bernstein, 1969; Bernstein & McAlister, 1976; Lichtenstein & Kentzer, 1971; Lichtenstein, Kentzer & Mees, 1968). This view was based upon the belief that research procedures which emphasize operational definitions, use well controlled hypothesis testing techniques and utilize behavior modification procedures, would ultimately provide valuable

practical and theoretical knowledge about smoking (Bernstein & McAlister, 1976), just as it has with other human behaviors (Bandura, 1969; Rimm & Masters, 1974). Most social learning approaches focus either upon (a) reducing the probability of smoking behavior, or (b) increasing the probability of an alternative non-smoking response. A few examples of each main social learning techniques are presented below.

Aversive control. One of the most common social learning techniques used to reduce the frequency of smoking behavior has been to utilize aversive stimuli, such as warm smoky air, electric shock, noise, or the aversive consequences produced by rapid smoking procedures. Aversive procedures usually involve pairing noxious stimuli of some sort with thoughts about, or actions associated with smoking. For example, when using electric shock as the aversive control devise, the shock paired with smoking, or sometimes self-administered by the client when merely thinking about smoking. Some of the studies which have utilized this technique include Best and Steffy (1971), Roy and Swillinger (1972), Russel (1971), Steffy, Meichenbaum, and Best (1970), and Whitman (1969).

Some researchers have investigated the use of satiation (rapid smoking) techniques (Claiborn, Lewis & Humble, 1972; Danaher, Lichtenstein & Sullivan, (in press); Lichtenstein, Harris, Brichler, Wahl & Schmahl, 1973; Marrone, Merksamer & Solzberg,(1970). The rapid smoking technique is a smoking control procedure that instructs the

participant to draw on a cigarette in a rapid (every six seconds) and continuous manner until further smoking cannot be tolerated.

Another aversive treatment approach is the warm smoky air method. This approach usually involves blowing warm, stale, smoky air into the face of the smoker while he is smoking his own brand of cigarettes. A few researchers who have investigated this approach are: Dawley, Ellithorpe, and Tretola (1976), Frank, Fried, and Ashem (1966), and Wilde (1965).

Stimulus Control. Simulus control tactics for reducing the probability of smoking behavior is another type of social learning approach. Stimulus control tactics are based on the assumption that smoking is associated with and prompted by environmental cues present prior to smoking, or while smoking occurs. Further, it is thought that since smoking usually takes place under a wide variety of circumstances, the number and extensiveness of these control cues or discriminative stimuli contribute to the habit (Bernstein & McAlister, (1976). Treatment usually involves a gradual elimination of smoking through programmed narrowing of the range of stimuli which are discriminative for smoking (Nolen, 1968). Stimulus control programs vary considerably with respect to how clearly they specify which environmental stimuli are to be detached from smoking. Some involve elimination of smoking from increasing numbers of specific situations, while others arrange only for non-smoking during certain periods of the day. Studies which utilize this type of procedure are Bernard and Efran (1972), Flaxman (1974), and Roberts (1969).

Reinforcement of Non-Smoking. The reinforcement of non-smoking is a social learning technique which seeks to eliminate smoking by strengthening other behaviors not involving, or perhaps incompatible with, smoking (Bernstein & McAlister, 1976). Positive reinforcement techniques usually employ either contingency contracting, coverant control, or both. A contingency contracting procedure would require the smoker to sign a contract stating, for example, that every hour he didn't smoke his wife would give him a dollar, and every time he smoked he would have to give a dollar to his wife or a charity.

A coverant control approach would attempt to reduce smoking by reinforcing the frequency of coverants ("Covert operants," or thoughts) incompatible with smoking (e.g., "smoking causes lung cancer"). The reinforcers could be presented by the experimenter, a social peer, or the subject himself. Some studies which use this approach are: Kentzer (1968), Lawson and May (1970), Rutner (1967) and Tooley and Prott (1967).

Drugs

The majority of the anti-smoking drugs which have been prescribed for would-be-quitters have either been designed to mimic the effects of nicotine or mitigate the physical and psychological consequences of smoking cessation (Bernstein & McAlister, 1976). The most widely used nicotinometic agent is lobeline sulphate. Researchers who have used lobeline sulphate to decrease smoking behavior have

included Ross (1966), Scott, Cox, MaClean, Prince and Southwell (1962), and White (1962).

Some other types of drugs which have been used are hydrooxyzine hydrochloride (Turle, 1958), meprobamate (Bartlett & Whiteheat, 1957); diozepan (trade name--Valium) and the stimulant methylphenidate (trade name--Ritalin) (Whitehead & Davies, 1964). The effectiveness of antismoking drugs in eliminating the smoking behavior of most subjects, as has been the majority of all treatment techniques, is usually only short-term and primarily a function of placebo and other non-specific effects associated with receiving medication rather than of specific drug characteristics (Bernstein, 1969; Schwartz, 1967).

The Effects of Nicotine on Behavior and EEG Patterns of Animals and Humans

Since the review by Silvette, Hoff, Larson and Haag (1962) on the actions of nicotine on the central nervous system, many research studies have been conducted which theorize that the nicotine supply a smoker obtains from the tobacco is a sufficient enough amount to be considered a major contributing factor for establishing and continuing the smoking habit (Jarvik, 1973; Jarvik, Glich & Nokomura, 1970).

Numerous researchers have indicated that nicotine affects both behavior and physiology of animals and humans (e.g., Goldstein & Nelson, 1974; Turner, 1971). Domino (1967) found that small doses of nicotine had no consistent effect on established conditioned pole-jump

behavior in the rat (less than 25 ug/Kg subcutaneously), or shock avoidance behavior in monkeys (40 ug/Kg intravenously). Acquisition of pole-jump behavior in the rat was slightly facilitated by 40 ug/Kg and depressed by 80 ug/Kg of nicotine, subcutaneously. Nicotine in doses above 250 ug/Kg consistently depressed established pole-jump avoidance behavior, producing a depression of avoidance, rather than of escape behavior.

Morrison and Lee (1968) showed that nicotine reduced the activity of spontaneously more active rats and increased that of less active animals. Furthermore, increased motor activity has been observed when the drug was injected in rats in the morning, which is their normal resting period (Bovet-Nitti & Oliverio, 1967). The same rats, when given nicotine in the night (active period) reduced their activity.

Armitage, Hall and Morrison (1968) indicated that nicotine increased the lever pressing activity in trained rats, and caused a change in EEG of cats indicative of cortical activation, which was considered consistent with the self-report of some smokers that inhalation of tobacco smoke caused them to be more alert and efficient. Nicotine administration before learning has been shown to improve the learning ability of rats and mice in several different tasks (Bovet, Bovet-Nitti & Oliverio, 1967; Bovet-Nitti, 1965; Garg & Holland, 1968).

Knapp and Domino (1962) first presented data which indicated that nicotine in small doses equivalent to those inhaled in tobacco smoke has a marked, but short-lasting stimulant effect on the brain

stem activating systems of various animals. This effect appeared on EEG readings within one minute after intravenous injections of 20 ug/Kg of nicotine and caused rapid activation of acute-midbrain-transected animals. Within four minutes, spindle bursts returned, often more prominent than before nicotine injection. Evidence that EEG activation involves an action of nicotine directly on the brain stem reticular formation has also been demonstrated by Domino (1967) and Kawamura and Domino (1969).

In essence, pharmacological studies on animals indicate that both nicotine and cigarette smoke produce different effects as a function of dosage, behavorial conditions, and the type of experimental animal (Armitage, Hall & Morrison, 1968; Barnes, 1966; Brown, 1966; Domino, 1967; Geller & Hartman, 1969; Hale, 1970; Schechter & Cook, 1976; Toda, 1976).

Effects of nicotine and tobacco smoking on human behavior and physiology is currently receiving attention from researchers in a variety of fields. This is easily exemplified by reviewing The Directory of On-Going Research in Smoking and Health, which is published by the United States Department of Health, Education and Welfare. However, the specific effects smoking has on physiology and especially on the EEG of humans is still a greatly debated issue. One group of researchers suggest that smoking acts as a stimulant and speeds up the brain wave activity, while another group of researchers suggest that smoking acts as a depressant and slows down brain wave activity. Several studies are presented in the following section which represent both sides of the argument.

The Effects of Nicotine and Smoking on the EEG Patterns of Humans

Brown (1974) conducted a study which sought to determine the relationship between degrees of smoking frequency and manifest EEG patterns. Brown investigated six different categories of smokers: (1) subjects who never smoked, (2) average smokers (3/4 to 1 1/4 packs/day), (3) heavy smokers (2 to 3 packs/day), (4) very heavy smokers (more than 3 packs/day), (5) former average smokers (an average smoker who has stopped smoking at least six months previously), and (6) former heavy smokers (a heavy smoker who had stopped smoking at least six months previously). Prior to presenting a summary of Brown's findings, however, it seems appropriate to provide a general discussion on brain waves for the reader who might have a limited background of EEG terminology. In general, brain waves are divided into four basic groups: Delta, Theta, Alpha and Beta. The individual group of brain waves are divided on the basis of their frequency of occurrence per second. For example: Delta brain waves are waves which occur at a frequency of 0-4 cycles per second (Hz); Theta waves occur at a 4-8 cycle per second frequency; Alpha waves occur at a 8-12 cycle per second frequency; and Beta waves occur at a 12-20 cycle per second frequency.

Although an individual's brain waves shift throughout the day from one frequency to another, EEG equipment provides researchers with the capability of determining which frequency of brain waves an individual is producing at any given time period. The equipment also allows us the ability to measure the strength of the brain

waves, or as referred to in EEG terminology, the amplitude of the brain waves (abbreviation for amplitude is uV). A summary of Brown's findings revealed that the EEG characteristics which provided the basis for significant discrimination between degrees of cigarette smoking frequency and non-smoking were: alpha frequency variations, amplitude of alpha, amplitude of beta, and the frequency of theta. More specifically, Brown noted the following individual brain waves differences between smokers and non-smokers.

Alpha Brain Waves

All active smoker subjects and the former heavy smoker group exhibited significantly higher frequencies of alpha than did the non-smokers and former average smoker groups. For average and for very heavy smokers, alpha frequency was significantly more variable. Variations appear to increase with increased frequency of alpha. The amplitude of alpha for the average smoker group was considerably larger than that of the never smoked group, whereas that for the very heavy smoker group was significantly smaller than the never smoked group. The percent time of alpha activity present in the EEG was similar for all groups, except for the very heavy smoker group, which contained approximately half the amount of alpha per unit of time.

Beta Brain Waves

According to Brown (1974), one of the chief characteristics of heavy-smoker EEG records is the extraordinary amount of rhythmic beta activity. A further difference between smoker and non-smoker groups in beta frequency range was found in the amplitude characteristics; the

amplitude of Beta for the smoker groups was nearly twice that for either the never smoked group or the former smoker groups. The total amount of beta activity present in the EEG (sum of both rhythmic and nonrhythmic) was similar for all groups except for the very heavy smoker group, which exhibited at least 50% more beta activity.

Theta Brain Waves

Although not statistically significant for individual group comparisons, Brown (1974) found that the trend of differences suggested that heavy smokers and very heavy smokers have slightly higher than average frequencies of theta. The distinguishing characteristic, however, is shown by the greater relative regularity of theta rhythm in all smoker and former smoker groups as compared to never smoked groups.

Brown's 1974 results confirmed and extended an earlier study (Brown, 1968) demonstrating marked differences in brain wave patterns between smokers and non-smokers. Brown reports that a clear cut relationship exists between patterns of EEG and degree of cigarette smoking frequency. EEG patterns of smokers and non-smokers differ for all the major characteristics, particularly in the frequency per unit of time and amplitude of both alpha and beta activity.

Brown reports the significance of the differences is more easily seen by comparing differences in EEG characteristics among three different categories of smokers (i.e., average smokers, very heavy smokers, and non-smokers). The EEGs of non-smokers appear to resemble the average EEGs of rest and relaxation characterized by predominantly slow wave activity varying between theta and alpha rhythms and with nonrhythmic activity appearing as a mixture of relatively slow waves

(Johnson, Lubin, Naitoh, Nute, & Austin, 1969; Lindsley, 1960; Volavka, Matousek, & Roubicek, 1967). The EEGs of very heavy smokers resemble the EEG patterns of intense activation characterized by desynchrony comprised of fast low-voltage activity (Daniel, 1965; Lindsley, 1960; Volavka et al., 1967) and the EEG patterns of average smokers contain the high frequency rhythmic activity suggestive of intermediate degrees of activation usually indicated by fast (13 to 20 Hz) rhythmic activity (Daniel, 1965; Gale, Dunkin & Coles, 1969; Volavka et al., 1967).

In general, Brown notes four outstanding and consistent EEG characteristics which are typical of individuals who have developed a habit of smoking cigarettes:

(1) increased frequency of alpha activity,

(2) increased amplitude of rhythmic beta activity between 13 and 20 Hz,

(3) less variation in frequency of theta (indicating predominantly high frequency theta), and

(4) greater abundance of identifiably different frequencies within the range between 3 and 20 Hz.

Thus, Brown suggests that smoking produces a tranquilizing effect on the human EEG or a general slowing down of the brain wave pattern.

Itil, Ulett, Hsu, Klingenberg, and Ulett (1971) also suggests a slowing down of brain waves as a result of smoking. They took 32 young chronic cigarette smokers and recorded their EEG's at the end of a 24 hour period of smoking deprivation and again after smoking three cigarettes. Using frequency and computer analysis, they present

an EEG change which indicates an increase in slow activity for ten minutes after smoking followed by a return to resting levels.

Phillips (1971) conducted a study in which he investigated the EEG changes associated with smoking in humans. His report compared computer analysis of EEG data recorded under both resting and work conditions following smoking to appropriate control data in six male twenty-five to thirty-five-year-old nurses. Following digitizing, a power spectral analysis was performed which revealed significant reductions in the peak alpha frequency component up to 20 minutes following smoking, during a visual task. Eyes-open resting data showed a similar but not significant loss after nine minutes. No indicators of increased fast activity was found, suggesting a general slowing down of the brain wave pattern.

In contradiction to the suggestion that smoking produces a tranquilizing effect on EEG, some studies have suggested a stimulating effect of smoking or in general a speeding up of the brain wave pattern.

Murphree, Pfeifer and Price (1967) demonstrated that drug effect on the central nervous system as seen in the EEG depend upon the subject's condition or state prior to administration. In the case of smoking, it was observed that a reflex effect in the EEG could occur after smoking but before any pharmacological effects can be seen in the blood, and that smoking seems to be a stimulant rather than a tranquilizer in most cases.

Ulett and Itil (1969) conducted a digital computer analysis of the EEG's of eight young heavy smoker males following 24 hours of the EEG's of eight young heavy smoker males following 24 hours of smoking deprivation. Results showed a significant increase in the slow frequencies which was reversed by the beginning of smoking. This study

suggests a general slowing down of the brain waves as a result of deprivation from smoking and a general speeding up of the brain wave pattern when smoking is reinstituted.

Hauser, Schwarz, Roth, and Bickford (1958) in a study of the effects of smoking on healthy young adults using the EEG and frequency analysis, found that 85% of smokers and 70% of non-smokers increased alpha frequencies by 1 to 2 Hz upon smoking. The change occurred early and was persistent. Four of five subjects smoked nicotine-free and cotton-simulated cigarettes and showed a similar increased alpha frequency. The authors of this study do not directly address the speeding up or slowing down of the brain wave activity issue, but it seems appropriate to assume that they are suggesting a speeding-up effect. Hauser, et al., also suggests that some of the effect smoking a cigarette has on the EEG pattern is directly related to the act of smoking and not the injesting of the nicotine, etc. into the body. Although this is entirely another issue it does seem to be an area which must be considered.

Biofeedback Approaches to the Treatment of Cigarette Smoking

Kamiya (1962) demonstrated that humans could be trained to control their EEG activity with biofeedback training. Since then many other investigators have replicated these findings (Beatty, 1972; Black, 1972; and Kamiya, 1969). To date, a review of the biofeedback literature indicates that only three attempts have been made to decrease the frequency of cigarette smoking via a biofeedback approach. The following is a brief summary of these studies. Havelick (1977) reported a case study in which he treated a 40 year old business

executive who reported having severe migraine headaches. The patient was unable to satisfactorily control the headaches medically (Valium and Cofergot) and over the years developed a dependency on Valium. It was decided to treat his migraine headaches with a combination of EMG and temperature biofeedback with in-vivo relaxation practice and generalization procedures. Elimination of headache activity was achieved in 16 sessions. Gradual withdrawal from Valium dependency was achieved by establishing weekly behavioral contracts for dosage reductions and instructing the patient to achieve "low arousal" (not defined in study) whenever withdrawal symptoms occurred. The final treatment goal was the elimination of cigarette smoking behavior. This goal was attempted only after other objectives described above were achieved. First the subject was instructed to wear a wrist golf stroke counter in order to establish baseline data of smoking frequency, as well as to provide information feedback. During this period, the subject was given EEG alpha training. After five sessions, the subject was able to sustain integrated EEG alpha levels of 20uV, as opposed to 12uV maximum with eyes closed during baseline. Tape recorded statements were turned on at a low volume level only while the subject was in an alpha condition. These statements included the following: "I can see myself working without cigarettes." "My lungs feel healthy." "I feel better without cigarettes." Havelick reported that within four months (16 sessions), his client had totally eliminated all headaches. Within 5½ months from the beginning of training, his client was not taking Valium, and within 6½ months his client had given up smoking, having previously smoked an average of 30 cigarettes per day.

Follow-up data indicated that these results were the same after a six month post-treatment period.

Turin and Nideffer (1974) reported a case study in which a patient with a headache history of 8-10 years was about to begin biofeedback training in an attempt to learn a finger warming strategy for the alleviation of her headaches. During the sixth week of baseline period, just prior to beginning actual training, the patient reported a sudden cessation of headache activity coincident with elimination of cigarette smoking. Subsequent monitoring of additional subjects finger temperatures prior to, during and after smoking demonstrated that decreased finger temperature is one effect associated with cigarette smoking. Thus, the cessation of smoking behavior in the patient described here was presumably associated with a spontaneous increase in finger temperature. Consistent with the results of increases in finger temperature through biofeedback training, the spontaneous increase found in this subject was associated with a dramatic reduction in headache activity.

This finding is interpreted as providing support for the notion that finger warming is indeed an active ingredient in the effectiveness of biofeedback based treatment of migraines. This support is especially important because the major studies in this area have employed virtually no controls for the effects of expectancy, impressive instrumentation, etc.

For four weeks the patient had continued to refrain from smoking. During this period her headaches decreased considerably, both in terms of actual number of headaches and amount of medication taken. Kothare (1975) combined yogic breathing, autohypnotic suggestions and

GSR-induced relaxation techniques to 1) modify excessive smoking behaviors in one group of 8 persons, and 2) control over-eating habits of 6 persons in the other group. A training program was set up to provide the participants with the use of relaxation as an active coping skill in the developing of self-control. Participants attended twice a week for 45 minutes each, for four weeks. As a result, six persons in the first group stopped smoking entirely; two persons reduced their smoking consumption of cigarettes considerably.

In the other group, all six persons altered their eating habits significantly towards desirable weight loss.

Statement of the Problem

Several reviews of research on the psychological treatments of cigarette smoking have indicated the need for additional treatment techniques that can produce long-term smoking reductions (Hunt & Bespolec, 1974; Hunt & Matarazzo, 1973; Hunt, 1973). Due to the lack of long-term reductions, considerable attention has been directed toward identification of physiological variables contributing to the maintenance of cigarette smoking (Stephens, 1977). To date, there appears to be some controversy as to the specific effects smoking a cigarette has on the EEG of humans. Brown (1974), Itil et al., (1971), Phillips (1971) suggest that smoking acts as a depressant and slows down brain wave activity, while Murphree et al., (1967), Ulett and Itil (1969), Hauser et al., (1958) suggest that smoking acts as a stimulant and speeds up the brain wave activity. The importance of clarifying these variables relates to the development of effective programs for the treatment of smoking behavior.

In summary, the questions are whether:

1) A biofeedback treatment technique can produce long-term reductions in smoking rate;
2) This study will provide additional research data to suggest that the smoking of a cigarette acts as a stimulant or a depressant on the EEG of humans; and,
3) The training of 8-12 Hz occipital EEG biofeedback will decrease smoking frequency.

The author hypothesized the following:

1) A biofeedback treatment technique will produce long-term reductions in smoking frequency;
2) Smoking a cigarette acts as a stimulant for some smokers, a depressant for other smokers, and possibly produces both stimulant and depressant effects for the same smoker on different smoking occasions; and,
3) The training of 8-12 Hz occipital EEG biofeedback training will decrease the frequency of smoking.

Purposes

This study attempted to identify the physiological variables which correlate with, and may possibly contribute to, the maintenance of cigarette smoking. Thus, it had two main objectives:

1) Determine the individual and simultaneous physiological changes i.e., EEG (brain wave patterns), EMG (muscle tension), EKG (heart rate), and hand skin temperature that occurred during and immediately after the smoking of one cigarette.
2) Determine what 8-12 Hz occipital EEG biofeedback training would have on smoking frequency.

individual is not really motivated to quit smoking. During the initial interview, each subject was first given a general outline of all procedures (Appendix B). Second, they were asked to complete a Smoker's Self-Testing Kit (Appendix C), which was developed by Daniel Horn, Ph.D., Director of the National Clearinghouse for Smoking and Health. The Self-Testing Kit was mainly administered to determine the degree of motivation the smoker had to quit smoking, however, the questionnaire also evaluated: 1) what effects the subject believed smoking had on him, 2) what the subject believed kept him smoking, and 3) how the world around him controlled his smoking frequency. Finally, they were asked to complete a general background questionnaire (Appendix D), which had been suggested for use in smoking cessation programs by the National Interagency Council on Smoking and Health.

The subjects who were selected from the group of volunteers were those six subjects who:

1) were either moderate or very heavy smokers,
2) scored nine or above on the Motivational Sub Test of the Smoker's Self-Testing Kit,
3) over the first three sessions of baseline had a mean percent 8-12 Hz activity of 95% or less,
4) indicated no health problems or current chronic conditions which their family physician felt would be negatively effected by the treatment phase of the study,
5) were not presently receiving any type of physician prescribed medication or on any other smoking programs, and

6) completed a general background questionnaire.

Subject permission and involvement were obtained in accordance with ethical guidelines for those subjects accepted into the study. A copy of the consent form can be found in Appendix E.

The following information was provided by the subject on the background questionnaire:

Subject 1 was 14 years old when he started smoking cigarettes regularly and he had been smoking for ten years. He had tried to quit smoking three or more times before, but had not been able to quit for more than a three month period. The methods he had used to attempt to quit smoking were drugstore remedies (Nicoban, Bantron, etc.). He reported that on the average he smoked 20 cigarettes per day. He was a college graduate and had a present occupation of Artist and part-time student. He mentioned that the only health problems he had was shortness of breath and that he smoked all brands of cigarettes, but presently was smoking Lucky Strike Filters.

Subject 2 was 15 years old when he started smoking cigarettes regularly and had been smoking for 7 years. He had tried to quit smoking once before, but had not been able to quit for longer than 24 hours. The method he had used to attempt to quit smoking was cold turkey. He reported that on the average he smoked 40-60 cigarettes per day. He was presently a Senior in college and part-time animal controller. He noted that he had no health problems and presently smoked Salem cigarettes.

Subject 3 was 18 years old when he started smoking cigarettes regularly and had been smoking for 12 years. He had tried to quit smoking once before, but had not been able to quit for longer than six days. He had not tried to use any particular method to try and quit. He reported that on the average he smoked 50 cigarettes per day. He was a college graduate and had a present occupation as a Sales Representative. He mentioned that the only health problem he had was a minor back problem and that he presently smoked Winstons.

Subject 4 was 16 years old when he started smoking cigarettes regularly and had been smoking for 13 years. He had tried to quit smoking once before, but had not been able to quit for more than 24 hours. The method he had used to attempt to quit smoking was a monetary contract. He reported that on the average he smoked 40 cigarettes per day. He was a college graduate and was presently working on a graduate degree. He mentioned that the only health problem he had was chronic bronchitis and that he presently smoked Kent Golden Lights.

Subject 5 was 31 years of age when he started smoking cigarettes regularly and had been smoking for 1 year. He had tried to quit smoking three or more times before, but had not been able to quit for more than six days. The methods he had used to attempt to quit smoking were drugstore remedies (Nicoban, Bantron, Water-pik filters, etc.). He reported that on the average he smoked 20 cigarettes per day. He was a college graduate and had a present occupation of Mathematician. He noted that he had no

health problems and presently smoked Merits.

Subject 6 was 19 years old when he started smoking cigarettes regularly and had been smoking for six years. He had tried to quit smoking three or more times before, but had never been able to quit for more than three months. The method he had used to attempt to quit smoking was a cold turkey approach. He reported that on the average he smoked 20-25 cigarettes per day. He was a college graduate and had a present occupation as a cabinetmaker. He mentioned that he had a history of asthma and was presently smoking Camel Filters.

Apparatus

The biofeedback equipment consisted of an Autogentic Systems, Inc., Feedback Encephalograph, Model 120a; a Feedback Electromyograph, Model 1100; a Feedback Skin Temperature Monitor, Model 1000; and a Feedback Electrocardiogram, Model FM-1100-4E. Other physiological recording equipment which was used, but was not designed to present feedback were: Narco Systems Physiograph (Model DMB 48), and a Healthtop blood pressure cuff and stethoscope.

The feedback encephalograph monitored the subject's brain wave activity and was equipped with adjustable frequency and amplitude filters. These filters were adjusted to define the EEG parameters that resulted in feedback (8-12 Hz with 0-80 Mv) and those parameters which did not result in feedback (12-20 Hz with 0-80 Mv and 4-8 Hz with 0-80 Mv). The feedback encephalograph had a meter which when switched to the main channel indicated the percentage of time the

subject's EEG was within the 8-12 Hz activity range and when switched to the auxiliary channel indicated the percentage of time the subject's EEG was within the 12-20 Hz activity range, for a period of 20 seconds. The feedback encephalograph also displayed by meters, the average frequency and the average amplitude of the subject's EEG within the 8-12 Hz range for the past accumulated 10 seconds of such activity.

The feedback myograph monitored the muscle activity and was equipped with adjustable scales and visual monitoring meter. These scales were adjusted at the beginning of each session for each subject to allow for observer's accurate monitoring of the visual meter. Although the feedback myograph was equipped to provide feedback for muscle activity, the unit was used only as a measuring device throughout this experiment.

Monitoring of hand skin temperature was the function of the feedback temperature unit. The temperature unit was also used as a measurement device and at no time throughout the experiment was the unit used to provide feedback to the subjects. The temperature unit was equipped with a control knob that could be adjusted to present a visual display of the subject's baseline skin temperature. As the visual display meter increased or decreased, observers could determine actual skin temperature.

The feedback electrocardiogram monitored the subject's heart beat and was equipped to transmit the heart beat through a portable transmitter, which was interfaced with a Narco Systems Physiograph. This allowed each subject's heart rate to be recorded on physiograph paper and later used to calculate the subject's heart rate per minute for each session.

The Healthtop cuff and stethoscope was used to monitor the subject's systolic and diastolic blood pressure at the beginning and end of each session. The blood pressure unit was equipped with an adjustable arm cuff and attached stethoscope. The unit also was equipped with a visual display meter.

Feedback Apparatus and Stimuli

The feedback encephalograph was used to provide feedback to the subjects. Each subject received "instantaneous" music feedback whenever his occipital EEG was within the 8-12 Hz activity range with 0-80 Mv. The music feedback was provided from an interfaced taped cassette recording of easy listening music from Andre Kostelanetz's album of the World's Greatest Love Songs #PG32002, which was manufactured by Columbia Records in 1973. The feedback was presented to the subjects during the training condition (see Table 1) for a minimum of eight sessions and a maximum of 21 sessions.

Experimental Setting

The individual sessions were conducted in a daily lit, 2.75 meters by 2.2 meters, moderately attenuated chamber in the Exceptional Child Center's Biofeedback Lab, Room 116C. The subject remained seated throughout the sessions in a comfortable recliner chair with the physiological electrodes attached. The electrode cables ran to an adjoining 2.75 meters by 2.75 meters room which housed the physiological equipment and the data recorders. A 30.48 cm x 38.10 cm one way mirror allowed the experimenter to view the subject.

Design

A multiple baseline design with replication across subjects was used (Baer, Wolf & Risley, 1968). The multiple baseline design requires continuous recording of the dependent variables of several subjects using baseline and experimental conditions. The independent variable is then introduced to each subject at different points in time during baseline. If changes in the dependent variables are due to the presentation of the independent variable, this will occur sequentially upon the presentation of the independent variable to each subject. The dependent variables recorded were: smoking frequency, EEG occipital amplitude, frequency, and percent of time within 8-12 Hz, EEG occipital percent of time within 4-8 Hz and 12-20 Hz with no amplitude criterion, EMG frontalis muscle tension, heart rate, and left hand skin temperature (See Appendices H through N). Note that subject six was unable to begin the study until day six. However, subject six still meets the requirements of the multiple baseline design.

The multiple baseline design is particularly useful when reversing the treatment conditions is undesirable (Baer, Wolf, & Risley, 1968). Other advantages of this design are: (a) all subjects are exposed to all treatment conditions, (b) a small subject sample can be used, (c) the possible effects of extraneous experimental variables such as time, placebo effects, attention, etc., can be controlled and (d) it applies to individual patients of concern to clinicians in the field.

Data Recorded

Four types of physiological data were recorded at 30 second intervals during each 30 minute session from each subject throughout all phases

of the experiment: (1) occipital EEG data, (2) frontalis EMG data, (3) heart rate data, and (4) hand skin temperature data. Blood pressure data were recorded by the experimenter at the beginning and end of each session. The physiological data were recorded by the experimenter or trained assistants on data sheets from visual inspection of equipment meters. The observers would always visually inspect the equipment meters in the following order: frontalis EMG data; skin temperature data; the percent time 8-12 Hz and 12-20 Hz activity; the mean frequency of 8-12 Hz activity; and the mean amplitude of 8-12 Hz activity. The data was recorded in the above manner to assure the experimenter that visual inspection of each physiological parameter was conducted at approximately every 30 second interval. Heart rate data was calculated at the conclusion of each session.

(1) Occipital EEG data. Occipital EEG data was recorded from electrode position O1 and T3, which were located over the left occipital cortex and temporal lobe (refer to Figure 1). A ground electrode was placed on the scalp over position T4 which is located over the right temporal lobe. The frequency filters on the feedback encephalograph were set at 8-12 Hz. The Amplitude filter was adjusted so that 8-12 Hz activity, between 0-80 MV in amplitude, was analyzed to compute percent of time, mean frequency, and mean amplitude. At 30 second intervals the experimenter's assistants recorded percent of time, mean frequency and mean amplitude of 8-12 Hz activity production during the processing periods by reading meters on the feedback encephalograph. The percent of time the subject's occipital EEG was within 8-12 Hz was computed for the past 20 seconds of real time.

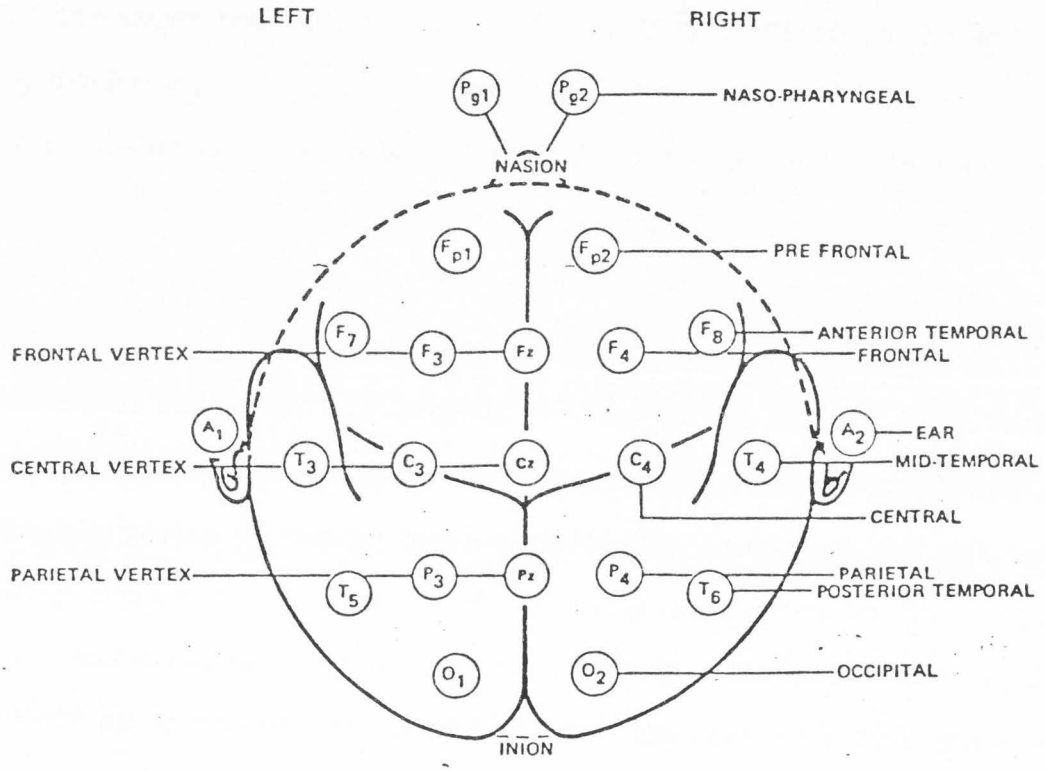


Figure 1. International 10-20 system for EEG electrode placements.

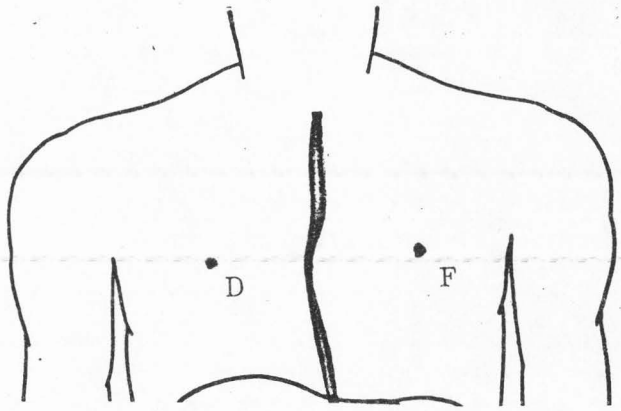


Figure 3. Electrode positions D and F for measuring heart rate.

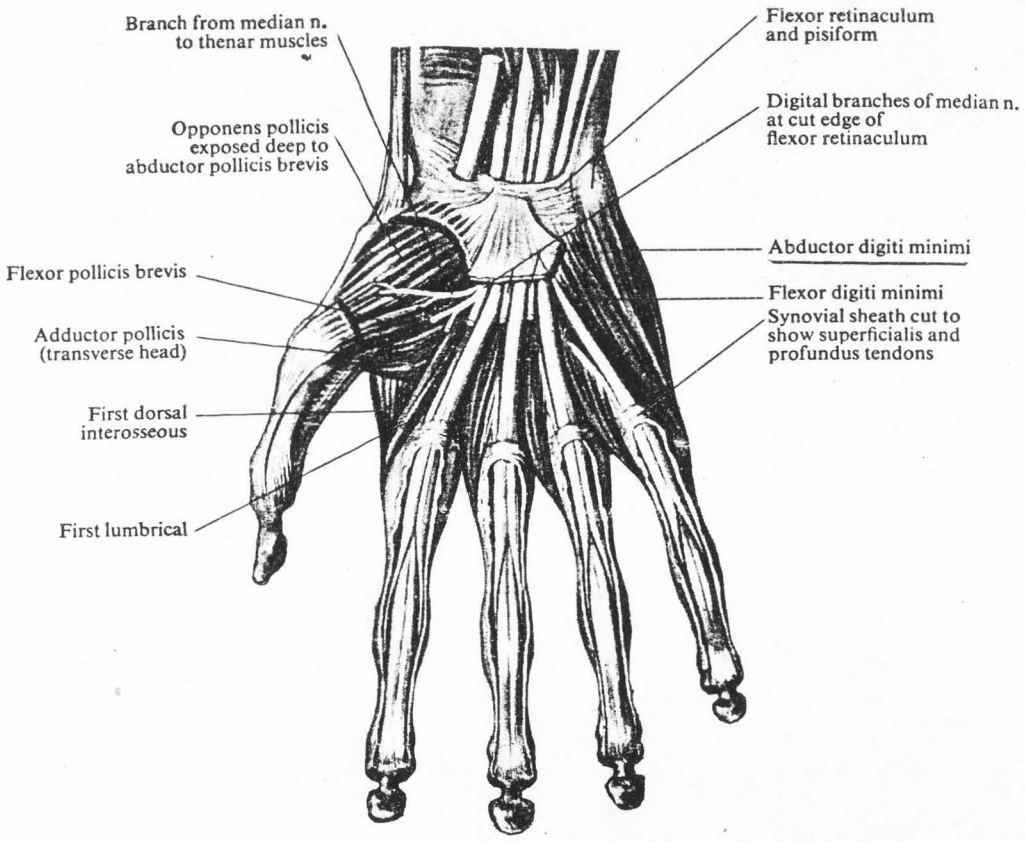


Figure 4. Muscles of the hand.

It is a sensitive method of measuring and quantifying muscular tension and relaxation. EMG frontalis muscle activity has been found to be useful indicator of general muscular tension and relaxaticn. Typically, EMG frontalis muscle activity below three microvolts is indicative of relaxed musculature (Stoyva & Budzynski, 1975). Skin temperature is used as an indice of autonomic nervous system functioning. Green (1972) has successfully demonstrated the relationship between relaxation and temperature rise using skin temperature as a measure. Hand skin temperature of 90° F. or higher is within a range considered to be indicative of autonomic relaxation. EEG brain wave activity is an indicator of cortical physiology. A predominant pattern of brain wave rhythms between 8-12 cycles per second (measured in Hertz units) is indicative of a relaxed cortical system, and between 12-20 Hz is indicative of a very active cortical system (Lawrence, 1972). The relationship between the amplitude of a particular brain wave pattern and the percent time production of the brain wave pattern has been a greatly debated issue. The research conducted to date on the amplitude/percent time relationship is still highly controversial and unsolved (Hardt and Kamiya, 1976; Plotkin, 1978).

(5) Blood pressure data. Blood pressure data was recorded by the experimenter from the subject's right arm at the beginning and end of each session. Systolic and diastolic recordings were made and recorded on the subject's data sheet.

Three types of behavioral data were recorded by the subject: (1) the Smoker's Self-Testing Kit was completed, (2) the general background questionnaire was completed, and (3) the subject's smoking

frequency inside and outside the experimental setting was measured. The first two types of behavioral data were gathered during the initial intake interview and serve merely as a screening device. The smoking frequency data served as a primary dependent variable.

(1) The Smoker's Self-Testing Kit. (See Appendix C). The Smoker's Self-Testing Kit was completed by each subject during the initial interview session. This self-testing kit was designed by Daniel Horn, Director of the National Clearinghouse for Smoking and Health. The test was designed to aid the smoker in answering some questions about his smoking habit. The questions, however, for the purpose of this study was used to aid the experimenter in deciding: (a) whether the smoker really wanted to quit smoking, (b) what the smoker knew about the effects of smoking on his health, (c) what kind of smoker he was (why he smokes), and (d) whether the smoker's environment helped or hindered him if he tried to stop. The degree of motivation the smoker had to quit smoking (Part A from above) was the primary purpose for the administration of the test. The subject's scores on the Smoker's Self-Testing Kit are presented in Appendix C-1.

(2) General background questionnaire. A general background questionnaire suggested for use by the National Interagency Council on Smoking and Health was administered to each subject during the initial intake interview (Appendix D). This background information questionnaire detected any health problems and gave the experimenter some data concerning how long the subject had been smoking, the number of times he had attempted to quit, etc.

(3) The subject's smoking frequency. Smoking frequency data were recorded outside the experiment proper on an hourly basis by each subject for the first three months of the study. The subjects were provided with recording cards which fit between the surrounding cellophane and the pack of the cigarettes. The subjects were required to record on the data card each time they smoked a cigarette. The daily smoking data cards were collected daily for the first three months of the study. Smoking frequency data recording was then switched to a one-day-a-week recording for the next three month follow-up. This procedure allowed for the fading out of any smoking decreases as a result of the recording procedure. One obvious disadvantage of utilizing a self-recording procedure to document smoking frequency change is that the person's self-reported data may be biased, inaccurate or falsified. There was no additional independent measure of the subject's smoking behavior recorded in this study. McFall (1978) reviews the pros and cons of utilizing self-report methods and discussed the problems associated with using additional unobtrusive naturalistic measures. The limitations of only using a self-report measure and the author's rationale for not implementing an additional measure of smoking frequency will be discussed in more detail in the Discussion Section of the paper. A copy of the smoking frequency data sheet can be found in Appendix F.

General Procedures

Throughout all phases of the experiment, each subject was requested to withhold from smoking for 1 hour prior to the lab sessions. Since the B phase of the study was designed to demonstrate the immediate

and after effects of smoking one cigarette on an individual's physiology, one hour of non-smoking prior to each lab session during all phases of the study was requested of each smoker to maximize the effects. Each subject was first seated in a recliner chair and then attached to the physiological equipment. Physiological data recording was conducted Monday through Friday at the same time of day for each subject, but at different times for the different subjects. Subjects were seen individually for 30 minutes of physiological data recording during each session. Prior to the beginning of the physiological data recording the subject was given approximately five to six minutes as an adaptation period (Meyers & Craighead, 1978). This was deemed necessary by the experimenter to allow for the heart rate, skin temperature, etc. to stabilize.

During all recording periods the experimenter and research assistants were located in the biofeedback equipment room, which was adjacent to the experimental chamber, in order to monitor the equipment. During all phases of the study, the subjects sat uninterrupted in a recliner chair with their eyes open. In view of the importance of maintaining constant alertness in EEG drug studies (Scott, Schwartz, Farrant, & Spiers, 1974), an alerting procedure similar to that of Volavka, Crown, Dornbush, Feldstein, and Fink (1973) and Knott and Venables (1977) was used throughout the study. Subjects were instructed to keep a button depressed on the arm of the chair, and whenever the button was released, a buzzer would sound.

Baseline A1

The previously noted smoking frequency data was recorded by all subjects at the beginning of the baseline period and continued throughout the study. The length of the baseline (A1) for each subject was determined by a combination of time and a data stability criterion. The subjects were required to have a minimum of three days separation prior to the implementing of a new phase. In addition, each subject's percent time within 8-12 Hz had to meet a stability criterion. The stability criterion was that each subject's mean percent time of 8-12 Hz activity for the last session had to be within one standard deviation of the mean of the last three sessions. For S2, S3, and S1 the lengths of the baseline were 4, 8, and 11 days respectively. For S4, S5, and S6 the lengths of the baseline were 3, 6, and 4 days respectively (see Appendix H and I). During the baseline A1 sessions each subject was fitted with the physiological electrodes, and told to sit quietly in the chair and rest with their eyes open. Subjects were numbered 1, 2, 3, 4, 5, and 6 after all the data was collected. The subjects were numbered in this manner to allow for easier graphical comparison between the smokers who quit smoking and those who did not.

Smoking B

During the smoking phase of the experiment, each subject was instructed to sit in a chair as in the Baseline A1 sessions. The first five minutes of physiological data recording for each smoking session was conducted as before. However, the next five minutes of the session, each subject smoked a cigarette of his choice, and was requested

to inhale the cigarette smoke at a rate which approximated the rate he inhaled in the natural environment. At the conclusion of the five minute smoking period the subject was told by the experimenter to extinguish the cigarette. The subject then remained seated for an additional 20 minutes of physiological data recording. The total number of smoking sessions per subject is shown in Table 1.

Baseline A2

Immediately after the smoking phase of the study, the subjects were again placed in a second baseline condition identical to that of the Baseline A1 condition. The number Baseline A2 sessions for each subject is shown in Table 1 (Appendix H through N demonstrates the Multiple Baseline Design Controls).

Feedback C: 8-12 Hz Occipital EEG Training

The number of 8-12 Hz feedback sessions for each subject is located in Table 1. Since the basic design of the study was a multiple baseline, the subjects were introduced to treatment after various amounts of physiological baseline (A1), (A2), and Smoking (B) data were collected: S2 after 10 sessions; S3 after 14 sessions; S1 after 17 sessions, S4 after 10 sessions, S5 after 14 sessions; and S6 after 10 sessions. In general during feedback sessions subjects were instructed to turn on the music feedback apparatus in the lab and then attempt to generalize their skill to the office, home, etc., and substitute the 8-12 Hz activity for a cigarette whenever they had the urge to smoke.

Table 1

Phase Sequence for Each Subject

Subject	(Type of Smoker)	Sessions	# of Days In Condition	Phase Condition
1	(Moderate)	1-11	11	Baseline A1
		12-14	3	Smoking B
(Total 9 days training)		15-17	3	Baseline A2
		18-25	8	Feedback C
		26-33	8	Fadeout D
3	(Heavy)	1-4	4	Baseline A1
		5-7	3	Smoking B
		8-10	3	Baseline A2
(Total 14 days training)		11-21	11	Feedback C
		22-29	8	Fadeout D
4	(Heavy)	1-8	8	Baseline A1
		9-11	3	Smoking B
		12-14	3	Baseline A2
(Total 20 days training)		15-32	8	Feedback C
		33-40	8	Fadeout D
4	(Heavy)	1-3	3	Baseline A1
		4-7	4	Smoking B
		8-10	3	Baseline A1
(Total 20 days training)		11-30	20	Feedback C
		31-38	8	Fadeout D

Table 1 (continued)

Subject	(Type of Smoker)	Sessions	# of Days In Condition	Phase Condition
5	(Moderate)	1-6	6	Baseline A1
		7-11	5	Smoking B
		12-14	3	Baseline A2
(Total 15 days training)		15-38	14	Feedback C
		29-32	4	Fadeout D (Death in family, had to leave town)
6	(Moderate)	6-9	4	Baseline A1
		10-14	5	Smoking B
		15-18	4	Baseline A2
(Total 21 days training)		19-38	20	Feedback C
		39-46	8	Fadeout D

Fadeout D: 8-12 Hz Occipital EEG Training

Fadeout Procedures

Each subject, upon completing the 8-12 Hz occipital training procedure, was exposed to an eight day fade out procedure. This phase of the experiment was designed to train the subjects to produce the desired 8-12 Hz physiological change without the aid of audible feedback for the entire session. The subjects were told that the feedback fadeout involved increasing the length of time at the end of the session during which the subject was to attempt to produce 8-12 Hz activity without receiving feedback. For example, each subject began the fadeout procedure by receiving six minutes of no feedback at the end of each session. Increases in the lengths of time no feedback was given were as follows:

Fadeout Phase	Minutes of No Feedback
Day 1	1
Day 2	8
Day 3	10
Day 4	12
Day 5	14
Day 6	16
Day 7	18
Day 8	20

It should be noted that on the final day of the fadeout (D) phase the subjects were exposed to 5 minutes of baseline, 5 minutes of feedback, and 20 minutes of no feedback, respectively.

Reliability

Physiological Data

Reliability of physiological data was established by having an independent student observer provide reliability checks on the data recorded by the experimenter or another assistant observer. The second observer sat in the chamber with the experimenter or another assistant observer and independently recorded physiological data displayed by the feedback equipment's meters (the experimenter was present during all observations to assure the observers recorded their data independently). For each subject, a minimum of two reliability checks were provided during each phase of the study. Therefore, a total of 60 reliability checks were taken across the six subjects. Of the total reliability checks taken, one reliability check was randomly selected from each phase for each subject to determine the average reliability taken across subjects. Therefore, a total of 30 reliability checks were utilized to determine the reliability of the data.

The average product moment correlation coefficients across subjects were computed for each physiological measure and served as an index or reliability. The coefficients were computed by randomly selecting one of the reliability sessions per phase per subject and comparing it to each of the sixty primary observers recordings. Thus, for each subject there was a total of five reliability calculations computed per physiological parameter. These coefficients were then averaged across all six subjects. The coefficients obtained for each measure were: percent time within 8-12 Hz activity = .9546, mean amplitude within 8-12 Hz activity = .9774, mean frequency within 8-12 Hz activity = .8800,

percent time within 12-20 Hz activity = .9560, microvolts of muscle tension = .9652, degrees of skin temperature = .9287, indicating a high level of inter-observer reliability. The extremely high correlation of coefficients are needed to indicate high levels of inter-observer reliability. Should below .85 coefficients be obtained the reliability of the data possibly should be questioned. The use of the product moment correlation to determine reliability of data can be questioned since great differences in the observations would be necessary to produce low coefficients. However, if extremely high levels of correlational coefficients are indicated the possibility of having unreliable data is relatively low.

RESULTS AND DISCUSSION

The results of the study are presented in three parts. The first part considers the subjects and their physiological parameters as a group, while the second part of the analysis focuses on changes in each individual subject's physiological responses to the various experimental phases. The third part of the analysis is presented in tabular form (Appendices H through O) to demonstrate that the Multiple Baseline Design requirements were met in this study. The results are presented in this manner for several reasons: (1) to aid in identifying the similar physiological changes which occur across subjects; (2) to emphasize the within-subject physiological differences across parameters that are associated with smoking; and (3) to aid in directing the construction of future hypothesis which attempted to explain decreases in frequency of smoking via a biofeedback approach. Both the group and the individual subject results are presented in a sequence to indicate: (1) the immediate effect of smoking a cigarette on physiology; (2) the after-effects of smoking a cigarette on physiology; (3) the 8-12 Hz occipital EEG training effect on physiology; (4) the 8-12 Hz occipital EEG training fade-out effect on physiology; and (5) the average number of cigarettes smoked per day, per phases.

Results are presented graphically in Figures 5 through 18 (beginning on page 92). The third part of the analysis is presented in tabular form (Appendices H through O) to demonstrate that the Multiple

Baseline Design requirements were met in the study. In these figures the data points shown represent the means of five-minute segments across the total number of sessions conducted during the first three phases (Baseline A1, Smoking B, and Baseline A2). For the remaining two phases (Feedback C and Fadeout D) the data points shown also represent the means of five-minute segments, however, the means are calculated from only the last three sessions for each phase. The last three sessions were used in phase C to present the subject's physiological parameters at that point in time during training in which the subject was producing 8-12 Hz activity at a stable percent of time during the session and when he has essentially learned the task as well as can be expected with the training technique utilized in this study (see Appendices O-1 through T to evaluate acquisition data and to determine the functional reinforcers during feedback). The last three sessions in phase D were used to represent more accurately the subject's final physiological parameters at the point in the Fadeout Phase where he received the least amount of feedback for 8-12 Hz activity. In other words, including all the sessions from phase C would have deflated the effects of training due to the subject's inability to produce high percentages of 8-12 Hz activity on the first few days of training. Likewise, including all the sessions from phase D would have inflated the effects of the fadeout procedure because the subject was not totally absent from feedback at all periods of the fadeout.

Another note should be mentioned prior to interpreting the data. When reading the graphs within each phase, it should be noted that the

data presented represent the average picture of a particular physiological parameter across the number of sessions conducted within that phase and that the gray area on the figures represents the variability across the sessions (standard deviation). Especially during the smoking phase (Phase B), it should be pointed out that this study investigated the effects of ingesting a small dosage of a drug (e.g., nicotine, etc.) on a particular physiological parameter for different subjects who have different body structures, weights, smoking histories, etc. Thus, it should be expected that for some subjects the effects of smoking on physiology will be short term and for some other subjects it will be long lasting, depending on the physical characteristics of the subject. This is the main reason why the data is presented in a manner to demonstrate the changes in physiology within sessions per phase (as is indicated in Figures 5 through 18) instead of across individual sessions as is presented in Appendix O-1 through T.

The following is a description of how the graphs are interpreted (the top panel of Figure 5 is used for reference, see page 93):

(1) The immediate effects of smoking a cigarette are determined by comparing the second data point, marked "X", in the Smoking (B) phase to the first data point in the Smoking (B) phase (reading the graphs from left to right) and to the second data points in both the Baseline (A1) phase and the Baseline (A2) phase.

(2) The after effects of smoking a cigarette are determined by comparing the last four data points in the Smoking (B) phase to the last four data points in the Baseline (A1) and Baseline (A2) phase.

(3) The 8-12 Hz occipital EEG activity training effect is determined by comparing the last 5 data points in Feedback (C) phase to the last 5 data points in the Baseline (A1) and Baseline (A2) phases.

(4) The 8-12 Hz occipital EEG training fadeout effect is determined by comparing the last three data points in the fadeout (D) phase to the last three data points in the Baselin (A1), Baselin (A2), and Feedback (C) phases. The last three data points of the Fadeout phase were used to determine whether or not the subject could increase his 8-12 Hz activity without the use of the biofeedback equipment. These data points were used because they represent the only data with no audible feedback presented.

(5) The average number of cigarettes smoked per day, per phase is determined by calculating the total number of cigarettes smoked while a subject was exposed to the conditions of a particular phase and then dividing that figure by the total number of days the subject was exposed to the phase condition. These data are shwon in Table 2, (refer to page 90).

Consistent Physiological Changes Across Subjects

In general, the smokers utilized in this study produced: decreases in 8-12 Hz activity (Ss 1, 2, 3, 4, 5, and 6) increases in 4-8 Hz activity (Ss 1, 2, 3, 4) and increases in heart rate during the five minute period while they actually smoked a cigarette (Ss 1, 2, 3, 4, 5, 6). Immediately after the smoking of a cigarette (i.e., within 20 minutes after extinguishing the cigarette) subjects 1, 2, 3, 5, and 6 demonstrated a continual increase in their heart rates while subjects 1, 4, 5, and 6 demonstrated a decrease in their skin temperature. The direction of

the brain wave patterns as a result of smoking for four possibly five of the smokers is in a slowing-down direction (Ss 1, 2, 3, 5, and possibly subject 4) while for subject 6 there is a speeding-up of the brain waves. The terms "slowing-down" and "speeding-up" are utilized to indicate a general shift of a subject's percent of time producing one range of Hz activity into another range of Hz activity. For example, subject 1 is approximately producing 8-12 Hz activity 75% of the time, 4-8 Hz activity 10% of the time, and 12-20 Hz activity 15% of the time during Baseline A1, and A2 periods. Now suppose the subject is asked to smoke a cigarette (B phase). After the smoking of a cigarette it is noted that his percent of time producing 8-12 Hz activity increases to 80% of the time while his 4-8 Hz activity decreases to 5% of the time and his 12-20 Hz activity remains relatively unchanged at 15% of the time (note that subject one's data indicates a slight decrease in 12-20 Hz activity, which could indicate that his brain wave pattern shifts more towards the 8-12 Hz range, subtracting from both the 12-20 Hz and 4-8 Hz range). This would indicate a "speeding-up" of the brain waves. If the subject's percent of time producing 4-8 Hz activity had increased and his 8-12 Hz activity had decreased, then the brain wave pattern would have been considered to have "slowed-down". It should be noted that there is a possibility that a subject could produce an initial "slowing-down" or "speeding-up" of the brain waves while smoking with a total reverse of the brain wave pattern's direction occuring immediately after the smoking of a cigarette (e.g., subjects 1 and 5). For the purpose of this study the direction of the brain wave pattern immediately after the smoking of a cigarette is considered the most important because

the data in this study indicates the immediate effect of smoking to be short lived. The author has also purposely not attempted to operationally define "slowing-down" or "speeding-up" of the brain waves for this study. The EEG data to date are not sophisticated enough to determine whether a 2% directional change in a brain wave pattern is relevant or if a 20% change is needed to produce significant changes in cigarette smoking frequency. Additional replication of this study will result in adding information to justify the construction of a data based operational definition of "slowing-down" and "speeding-up". In addition, it should be noted that the pre-post blood pressure data per phase revealed no significant changes in blood pressure as a result of smoking or treatment and thus will not be discussed. The data are, however, available in Appendix G.

In reviewing the group changes more specifically, the data indicated that of the six smokers, subjects demonstrated the following physiological changes while actually smoking one cigarette. It should be pointed out that the immediate effect of smoking a cigarette is determined by comparing the second data point, marked "X", in the Smoking (B) phase to the first data point in the Smoking (B) phase to the second data points in both the Baseline (A1) phase and the Baseline (A2) phase. The immediate effect of smoking a cigarette data indicated that the percent of time within the 8-12 Hz activity range decreased (Figure 5, Subjects 1, 2, 3, 4, 5, and 6) and that the percent of time within the 4-8 Hz activity range increased (Figure 8, Subjects 1, 2, 3 and 4).

Immediately after the smoking of one cigarette (the after-effects of smoking a cigarette is determined by comparing the last four data points in the Smoking (B) phase to the last four data points in the Baseline (A1) and Baseline (A2) phases), five of the smokers increased their heart rates (Figure 11, Subjects 1, 2, 3, 5, and 6) and four subjects showed a decrease in their skin temperature (Figure 12, Subjects 1, 4, 5, and 6). There weren't any specific consistent Hz brain wave changes across the subjects. For example: Subjects 2, 3, and 4 increased their 4-8 Hz activity (Figure 8); Subjects 1 and 5 increased their 8-12 Hz activity (Figure 5), and Subject 6 increased his 12-20 Hz activity (Figure 9). However, there was a general slowing down in the cycles per second level for four possibly five of the smokers (Subjects 1, 2, 3, 5, and possibly 4) with one subject (Subject 6) displaying a speeding-up pattern in his brain waves (Figure 13 through 18, note that all EEG graphs will be discussed in more detail in the individual subject data which follows).

During the 8-12 Hz occipital EEG training feedback Phase (C), when the smokers were given music feedback whenever they produced 8-12 Hz occipital EEG activity, four of the six smokers learned to increase the percent of time spent producing 8-12 Hz activity compared to their baseline levels (Figure 5, Phase (C), Subjects 1, 2, 5, and 6). (Note that the 8-12 Hz occipital EEG training effect is determined by comparing the last five data points in the Baseline (A1) and Baseline (A2) phases to the last five data points in the feedback (C) phase).

During the 8-12 Hz occipital EEG training Fadeout Phase (D) two of these four smokers were able to continue producing high levels

of 8-12 Hz activity without the use of the biofeedback equipment (Figure 5, Phase D, Subjects 1 and 2). (The 8-12 Hz occipital EEG training fadeout effect is determined by comparing the last three data points in the Fadeout (D) phase to the last three data points on the Baseline (A1), Baseline (A2) and Fadeout (C) phases.) Subject Five's fadeout data also appears to indicate that he was able to produce slightly higher than Baseline (A1) or (A2) levels of 8-12 Hz activity without the use of the biofeedback equipment (Figure 5, Subject 5, Phase D) however, this level is extremely inflated due to the subject's receiving only four days of fadeout training. Due to personal family problems the subject was forced to leave the experiment prematurely and thus, his fadeout data only reflects a time period of approximately ten minutes at the end of the session at which time he was receiving no music feedback. A more representative indication of subject five's ability to produce 8-12 Hz activity without the use of the biofeedback equipment is located in the first 5-minute baseline data point of the phase (Figure 5, Subject 5, Phase D). It is concluded from this information that Subject Five had not acquired the ability to control his 8-12 Hz activity level without the use of the biofeedback equipment.

In summary, the data indicated that of the six subjects, Subjects 1 and 2 were the only two who were able to produce a higher percent of time of 8-12 Hz occipital EEG activity at the conclusion of the study, as compared to their previously demonstrated baseline levels. These smokers (Subjects 1 and 2) had quit smoking cigarettes completely at the end of the six-month follow-up period (Table 2). These two smokers were contacted by phone at the eight-month follow-up period

and reported that they were still abstaining from any cigarette smoking. The four smokers who could not increase their 8-12 Hz waves without the use of the biofeedback equipment only decreased their frequency of cigarette smoking at the 6 month follow-up period as follows: Subject 3, from 38 to 15 cigarettes smoked per day; Subject 4, from 50 to 44 cigarettes smoked per day; Subject 5, from 18 to 15 cigarettes smoked per day; and Subject 6, from 17 to 10 cigarettes smoked per day.

As to why Subjects 3 and 6 showed a moderate decrease in their smoking frequency, the author can only speculate that it was due to some specific individual physiological change that occurred as a result of 8-12 Hz activity training and fadeout procedures or that the decrease was due to some other variable such as: the self-recording procedures (possibly subject 5); the subject's expectation to decrease smoking; the placebo effect of seeing all the biofeedback equipment; the subject's smoking history or whatever.

One additional point concerning the group data should be brought up prior to proceeding on to the individual subject data. Across the majority of the smokers it was noted that there were extreme amounts of variability (Standard Deviation) in some parameters, especially the muscle tension data. It was concluded that this was due to the subject's eye movement as a result of allowing him to keep his eyes open throughout the study.

Individual Subject's Physiological Changes

The individual subject's physiological changes during each phase are presented for four reasons: (1) to present information concerning

how smoking a cigarette affects physiological parameters differently for different subjects; (2) to present information concerning how the training of 8-12 Hz activity affects other physiological parameters; (3) to aid in analyzing possible reasons for why some smokers decreased the frequency of cigarette smoking more than other smokers; and (4) to aid in directing the construction of new hypotheses which attempt to explain decreases in the frequency of cigarette smoking via a bio-feedback approach.

The individual subject's physiological data to be discussed are presented in Figures 13 through 18 (refer to page 109). The data in these are taken from the data in Figures 5 through 12 and are rearranged to simplify comparisons across physiological parameters within a subject. Due to the enormous amount of data only the individual subject's physiological parameters which demonstrate some apparent significant change as a result of smoking or training will be discussed. Those parameters which fluctuate only a small degree, do not fluctuate at all, or have unstable baselines will usually not be discussed. The direction and degree of change for each physiological parameter during and immediately after the smoking of a cigarette were determined by making comparisons to the first five-minute baseline data point in the Smoking (B) Phase and to the subject's overall physiological patterns demonstrated in the Baseline (A1) and (A2) Phases. The direction and degree of change in the physiological parameters during the Feedback (C) and Fadeout (D) Phases were determined by making comparisons to the mean levels in the Baseline (A1) and (A2) Phases. For the EEG percent Hz activity data a general criteria for discussing the degree

change will be as follows: 2% to 4% change equals a slight change; 5% to 9% change equals a moderate change; and, a 10% or greater change equals a large change. If there appears to be either an increase or decrease in the data, but the baseline data graphs demonstrate unstability then the term "increase" or "decrease" will be used independent of slight, moderate, or large. There is no data available to justify the use of operationally defining these degrees of changes as slight, moderate or large, however, it does clarify the use of terms for this study. The degree of change for the heart rate, skin temperature, amplitude, and frequency will be presented only in terms of increase, decrease, or no change.

Subject One (Figure 13)

While smoking a cigarette (represented graphically by point "X" in Phase B) Subject One produced an increase in 4-8 Hz activity (Panel 2, Phase B), a moderate decrease in 8-12 Hz activity (Panel 1, Phase B) and an increase in 12-20 Hz activity (Panel 3, Phase B), compared to the second data points in Baseline A(1) and A(2) phases and the first data point in the B Phase. Both the subject's muscle tension (Panel 4, Phase B) and heart rate (Panel 5, Phase B) increase while his amplitude and frequency of 8-12 Hz activity either did not change or had unstable baselines which hindered interpreting the data.

Within a 20 minute time span after smoking a cigarette (which is represented graphically by the four data points which follow the "X" data point in Phase B) Subject One produced: (1) an initial large increase in 8-12 Hz activity which lasted for approximately a

ten-minute period and then recovered towards its five-minute baseline level (Panel 1, Phase B); (2) an initial large decrease in 12-20 Hz activity followed by a return towards baseline (Panel 3, Phase B); (3) a slight decrease in 4-8 Hz activity which also returned towards baseline levels as the effect of smoking wore off (Panel 2, Phase B): and (4) an increase in the frequency of 8-12 Hz activity which remained above baseline levels throughout the session (Panel 8, Phase B). Subject One's heart rate increased and remained above Baseline (A1) and (A2) levels throughout the remaining 20 minutes of the session (Panel 5, Phase B). His skin temperature initially decreased and remained lower than both Baseline (A1) and (A2) Phases (Panel 6, Phase B). Subject One's demonstrated EEG pattern shift, which occurred immediately after the smoking of a cigarette, suggests that he neither smoked to speed up his brain waves or slow them down. What is concluded is that smoking merely increased Subject One's 8-12 Hz brain waves, which is sometimes referred to in the literature as increasing one's alpha-state, and which is associated with an awake, mentally relaxed state.

During the 8-12 Hz Feedback (C) Phase of the experiment, (compare the last five data points in the Feedback (C) Phase to the last five data points in the Baseline (A1) and (A2) Phases), Subject One showed the following changes: (1) percent time 8-12 Hz activity moderately increased and stabalized (Panel 1, Phase C); (2) percent time 4-8 Hz activity remained unchanged (Panel 2, Phase C): (3) percent time 12-20 Hz activity moderately decreased and stabalized (Panel 3, Phase C); (4) muscle tension remained unchanged (Panel 4, Phase C): (5) heart rate decreased (Panel 6, Phase C); (6) skin temperature increased

(Panel 6, Phase C); (7) amplitude of 8-12 Hz activity remained unchanged (Panel 7, Phase C); and (8) frequency of 8-12 Hz activity decreased (Panel 8, Phase C). Thus, during the Feedback Training Phase (C) Subject One was successful in increasing his 8-12 Hz activity when feedback was provided. In conjunction with his increase in 8-12 Hz activity Subject One's heart rate decreased, his skin temperature increased, and his 12-20 Hz activity decreased. All of these physiological changes are in the direction which is typically assumed to demonstrate a more relaxed physiological pattern.

Upon conclusion of the 8-12 Hz occipital EEG training Fadeout Phase (D), (compare the last three data points in the Fadeout (D) Phase to the last three data points in the Baseline (A1), (A2), and Feedback (C) Phases) Subject One's 8-12 Hz activity showed a large increase (Panel 1, Phase D), 12-20 Hz activity (Panel 3, Phase D) demonstrated a large decrease and 4-8 Hz activity (Panel 2, Phase D) slightly decreased, Subject One's heart rate increased above Baseline (A1) and (A2) levels (Panel 5, Phase D). Thus, Subject One's (Fadeout D) data indicated that he had learned to increase his percent time of 8-12 Hz activity without the use of the biofeedback equipment. In conjunction with a large increase in 8-12 Hz activity Subject One produced large decreases in his 12-20 Hz activity and increases in his heart rate. It should be noted that these three physiological changes which occurred when Subject One increased his 8-12 Hz activity also changed in the same direction after Subject One finished the smoking of a cigarette.

A look at Subject One's frequency of cigarette smoking (Table 2) indicates that he abstained from smoking at: the 3 month follow-up

point, during which time he recorded his daily cigarette consumption seven days a week; the 6-month follow-up point, during which time he recorded his cigarette consumption for one, randomly chosen day a week; and, at the 8-month follow-up point, when he discontinued the use of any form of self-recording procedure. The 8-month follow-up was conducted by phone.

Subject Two (Figure 14) (refer to page 111)

During the actual smoking of a cigarette, Subject Two produced: a large decrease in 8-12 Hz activity (Panel 1, Phase B); a large increase in 4-8 Hz activity (Panel 2, Phase B); no change in 12-20 Hz activity (Panel 3, Phase B) and, an increase in heart rate (Panel 5, Phase B). Both frequency of 8-12 Hz activity (Panel 8, Phase B) and skin temperature (Panel 6, Phase B) did not change while the amplitude of 8-12 Hz activity baseline data was not stable enough to permit clear interpretation (Panel 7, Phase B). Muscle tension (Panel 4, Phase B) possibly increased, but the variability was so great during this Phase (B) and the following Baseline Phase (A2) that little can be concluded.

In the next 20 minutes after he smoked a cigarette, Subject Two produced: (1) a slight decrease in 8-12 Hz activity, which remained below the mean Baseline (A1) and (A2) levels throughout the session (Panel 1, Phase B); (2) a moderate increase in 4-8 Hz activity, which remained above all baseline levels (Panel 2, Phase B); and (3) a slight decrease in 12-20 activity, which also remained below the mean Baseline (A1) and (A2) levels (Panel 3, Phase B). His heart rate initially increased, but then returned to baseline levels (Panel 5, Phase B)

and his skin temperature increased and remained above baseline levels. Subject Two's amplitude of 8-12 Hz activity initially decreased and then returned towards Baseline (A1) and (A2) levels (Panel 7, Phase B). The frequency of 8-12 Hz activity remained unchanged (Panel 8, Phase B) and muscle tension's standard deviation again varied to an extent which made the data uninterpretable (Panel 4, Phase B). Thus, Subject Two's data indicated that when he smoked a cigarette and immediately after the smoking of a cigarette his brain wave activity slowed down from that of 8-12 Hz activity to that of 4-8 Hz activity. His heart rate speeded up and his skin temperature increased.

During the 8-12 Hz occipital EEG Feedback Phase of the experiment Subject Two's: percent time of 8-12 Hz activity largely increased (Panel 1, Phase C); percent time 4-8 Hz activity slightly decreased (Panel 2, Phase C); percent time 12-20 Hz activity slightly decreased (Panel 3, Phase C); and, muscle tension was again quite variable, however, there appeared to be a decrease in the mean level (Panel 4, Phase C). Both heart rate (Panel 5, Phase C) and skin temperature showed a large increase (Panel 6, Phase C). Amplitude of 8-12 Hz activity increased (Panel 7, Phase C), and frequency of 8-12 Hz activity showed a decrease (Panel 8, Phase C).

Thus Subject Two was successful in increasing his 8-12 Hz activity when feedback was provided. Remarkably, when he increased the percent of time of 8-12 Hz activity he also altered all of the other physiological parameters, most of which were in the direction of producing a more relaxed state (i.e., muscle tension decrease, skin temperature increase, 8-12 Hz activity increase, etc.).

Upon conclusion of the 8-12 Hz occipital EEG training Fadeout Phase (D) Subject Two maintained a moderately high level of 8-12 Hz activity above Baseline (A1) and (A2) levels (Panel 1, Phase D); his heart rate (Panel 5, Phase D), skin temperature (Panel 6, Phase D), and amplitude of 8-12 Hz activity (Panel 7, Phase D) remained above Baseline (A1) and (A2) levels. He maintained low levels of 4-8 Hz activity (Panel 2, Phase D), 12-20 Hz activity (Panel 9, Phase D), muscle tension (Panel 4, Phase D), and frequency of 8-12 Hz activity (Panel 8, Phase D).

Therefore, Subject Two's Fadeout (D) Phase data indicated that he had acquired the ability to increase his 8-12 Hz activity without the use of biofeedback. When he increased his 8-12 Hz activity without the use of biofeedback he again simultaneously altered all of the other physiological parameters, the majority of which were in the direction which also is suggestive of maintaining a relaxed state. It should also be noted that three of these alterations in physiology were in the same direction that was recorded when he smoked a cigarette (i.e., 12-20 Hz decreased, heart rate increased, and skin temperature increased).

Subject Two's smoking data indicated that he had quit smoking at the 3-month, 6-month, and 8-month follow-up periods. However, there is some questions as to why Subject Two quit smoking after he was provided with 8-12 Hz feedback training, when in fact, he produced a 4-8 Hz activity increase after smoking a cigarette. Although he did not produce an increase in 8-12 Hz activity as a result of smoking, the increase he produced in 4-8 Hz activity does suggest a general slowing down of his brain waves.

Subject Three (Figure 15) (refer to page 113)

While Subject Three was smoking a cigarette he produced a moderate increase in 4-8 Hz activity (Panel 2, Phase B), a slight increase in 12-20 Hz activity (Panel 3, Phase B), and an increase in heart rate (Panel 5, Phase B). Subject Three's 8-12 Hz activity decreased largely (Panel 1, Phase B) and his amplitude of 8-12 Hz activity (Panel 7, Phase B) decreased. Subject Three's mean skin temperature was above Baseline (A1) and (A2) levels, however, the initial five minute baseline period, which was recorded at the beginning of the Smoking Phase, was also above the first five minute data point of the Baseline (A1) and (A2) Phases, so directional interpretations are not possible. There was no apparent change in Frequency of 8-12 Hz activity, and muscle tension data again demonstrated some degree of variability, so directional data interpretation were not possible.

Shortly after smoking a cigarette Subject Three produced: (1) an initial slight increase in 12-20 Hz activity (Panel 3, Phase B) and a moderate increase in 4-8 Hz activity (Panel 2, Phase B) which lasted for approximately five to ten minutes and then demonstrated a recovery towards baseline; (2) an increase in heart rate, which remained throughout the session (Panel 5, Phase B); (3) an increase in skin temperature, which remained throughout the session (Panel 6, Phase B): (4) an increase in the average frequency of 8-12 Hz activity, which was maintained for the remainder of the session, but which appeared to recover slightly towards baseline (Panel 8, Phase B); and (5) an initial moderate decrease in 8-12 Hz activity with a recovery towards baseline immediately after the smoking of a cigarette was completed (Panel 1,

Phase B). The subject's amplitude of 8-12 Hz activity Baseline (A2) Phase was not stable enough for clear interpretation of the data.

Thus, when Subject Three smoked a cigarette and for a short period thereafter, his brain wave activity shifted out of the 8-12 Hz range and into the 4-8 Hz and 12-20 Hz range with the predominate pattern shifting towards the 4-8 Hz range indicating a slowing down of his brain waves. His heart rate, skin temperature, and frequency of 8-12 Hz activity also increased shortly after the cigarette had been smoked.

Upon conclusion of the training Fadeout Phase (D), Subject Three's brain waves had not changed as compared to the Baseline (A1) and (A2) Phases (Panels 1, 2, and 3, Phase D), but his muscle tension had decreased (Panel 4, Phase D) and his heart rate (Panel 5, Phase D), skin temperature (Panel 6, Phase D), frequency of 8-12 Hz activity (Panel 8, Phase D) and amplitude of 8-12 Hz activity (Panel 7, Phase D) had increased.

It should be noted that the increase in his heart rate and in the frequency of 8-12 Hz activity paralleled both of those changes in the same physiological parameters recorded shortly after he had smoked a cigarette. His amplitude of 8-12 Hz activity could possibly have increased, but the elevated (A2) Baseline Phase as compared to the (A1) Phases makes the interpretation of the increased level in Fadeout Phase difficult. Thus, Subject Three did not learn to increase his 8-12 Hz activity, (but when he attempted to do so) he produced changes in three other parameters which changed in the same direction as did his physiological parameters when he had finished smoking.

Examination of Subject Three's cigarette smoking data indicated that his frequency had decreased by 61% at the completion of the study, from 38 to 15 cigarettes per day.

Subject Four (Figure 16) (refer to page 115)

While smoking a cigarette Subject Four produced a moderate increase in 4-8 Hz activity (Panel 2, Phase B) and a slight increase in 12-20 Hz activity (Panel 3, Phase B). This subject's skin temperature (Panel 6, Phase B) decreased and his percent of time producing 8-12 Hz activity moderately decreased (Panel 1, Phase B). His heart rate (Panel 5, Phase B) and frequency of 8-12 Hz activity (Panel 8, Phase B) did not change during smoking. Subject Four's amplitude of 8-12 Hz activity in the Baseline (A2) Phase did not return to the Baseline (A1) Phase level so data interpretation was impossible (Panel 7, Phase B). The subject's muscle tension increased, but the variability of this measure was considerable (Panel 4, Phase B).

Thus, Subject Four's data indicated that when he is smoking a cigarette his brain wave pattern shifts into the 12-20 Hz range and 4-8 Hz range, with the majority of the shift in the 4-8 Hz range.

Upon completion of smoking a cigarette and within 20 minutes after smoking, Subject Four produced initial slight decreases in: 8-12 Hz activity (Panel 1, Phase B); muscle tension (Panel 4, Phase B); and skin temperature (Panel 6, Phase B) as compared to his first five minute baseline data point of the phase. All of these parameters recovered however towards their initial Baseline levels by the end of the session. Although the Baseline (A2) Phase of the 4-8 Hz activity data (Panel 2, Phase B) had not recovered to the Baseline

(A1) Phase level, it is suggested that Subject Four's activity initially increased as compared to the first data part of the Baseline and the overall level was higher than in the baseline (A1) phase (Panel 2, Phase B). His heart rate appeared to have increased (Panel 5, Phase B) while his 12-20 Hz activity (Panel 3, Phase B) and frequency of 8-12 Hz activity had not changed (Panel 8, Phase B).

The 8-12 Hz training data (Panel 1, Phase C) indicated that Subject Four did not learn to increase his 8-12 Hz activity and had not altered his brain wave activity in any distinguishable manner with the exception of a large increase in amplitude of 8-12 Hz activity (Panel 7, Phase C) and a slight decrease in his frequency of 8-12 Hz activity (Panel 8, Phase C). Correlated with these changes is a sharp decrease in his muscle tension (Panel 8, Phase C) and an increase in his heart rate (Panel 5, Phase C).

Upon completion of the 8-12 Hz occipital EEG training Fadeout Phase Subject Four's: (1) 4-8 Hz activity showed a dramatic increase (Panel 2, Phase D); (2) heart rate increased (Panel 5, Phase D); amplitude of 8-12 Hz activity has increased (Panel 7, Phase D); 8-12 Hz activity had decreased (Panel 1, Phase D); 12-20 Hz activity has decreased (Panel 3, Phase D); and the average frequency of 8-12 Hz activity has slightly decreased (Panel 8, Phase D). Due to the sleep alert sounding twice while Subject Four was in Phase D of the experiment these changes in physiology were probably due to the subject's falling asleep.

In general, immediately after the smoking of a cigarette, Subject Four's brain wave shift is exclusively in the 4-8 Hz activity range.

Upon completion of the Fadeout Phase, Subject Four's data indicate that he has not learned to increase his 8-12 Hz activity during training and he has probably fallen asleep occasionally during the Fadeout Phase. It is interesting to note that two physiological parameters changed in the same direction during training fadeout (Phase D) as did his physiological parameters shortly after he completed the smoking of a cigarette (8-12 Hz activity decrease and 4-8 Hz activity increase). Subject Four's frequency of cigarette smoking had decreased 12% at the completion of the study, from 50 to 44 cigarettes per day.

Subject Five (Figure 17) (refer to page 117)

During the smoking of a cigarette Subject Five produced: increases in heart rate (Panel 5, Phase B), amplitude of 8-12 Hz activity (Panel 7, Phase B), and in muscle tension (Panel 4, Phase B). He also showed a slight decrease in 8-12 Hz activity (Panel 1, Phase B); and, what appeared to be an increase in the skin temperature, as compared to the initial five minute baseline period within the smoking phase, but an overall decrease in skin temperature as compared to the Baseline (A1) and (A2) levels (Panel 6, Phase B).

Shortly after the smoking of a cigarette Subject Five produced: (1) a continuous large increase in 8-12 Hz activity (Panel 1, Phase B); (2) an increase in heart rate, which returned towards baseline levels (Panel 5, Phase B); (3) a continual large decrease in 4-8 Hz activity (Panel 2, Phase B); (4) a slight decrease in 12-20 Hz activity with a recovery towards baseline at the end of the session (Panel 2, Phase B); (5) a clear decrease in muscle tension, which also recovers

towards baseline by the end of the session (Panel 5, Phase B); (6) a decrease in skin temperature (Panel 6, Phase B); and (7) an immediate short period increase in the amplitude of 8-12 Hz activity, which recovers approximately ten minutes after the smoking of a cigarette and then proceeds to go below baseline levels (Panel 7, Phase B).

During the training phase, Subject Five demonstrated a moderate increase in his 8-12 Hz activity over baseline levels (Panel 1, Phase C). His muscle tension decreased (Panel 4, Phase C) and his heart rate increased (Panel 5, Phase C), while his skin temperature increased above his Baseline (A2) Phase, but only slightly above his (A1) Phase (Panel 6, Phase C).

Upon conclusion of the 8-12 Hz occipital EEG training Fadeout Phase Subject Five's: 8-12 Hz activity had increased (Panel 1, Phase D); his heart rate had increased (Panel 5, Phase D); and his amplitude of 8-12 Hz activity had increased slightly (Panel 7, Phase D). Note however, that Subject Five was not able to complete the last four days of the Fadeout Phase. Thus, it was concluded that these findings were inflated (e.g., the data suggests that Subject Five could control his 8-12 Hz activity without feedback, when in fact he could not) due to the fact that the most time that he received no feedback was 12 minutes.

During the training phase, Subject Five learned to increase his 8-12 Hz activity during training, however, it is questionable whether he still retained the ability to do so during the Fadeout procedure. It is interesting to note that two physiological parameters changed in the same direction shortly after he had completed the smoking of a cigarette (8-12 Hz activity increased and heart rate increased) as

they did when he increased his 8-12 Hz activity. The author is cautious in interpreting these results due to the lack of a completed Fadeout Procedure. Subject Five's smoking frequency data indicated that he had decreased his smoking frequency 17% at the completion of the follow-up period, from 18 to 15 cigarettes per day.

Subject Six (Figure 18) (refer to page 119)

While Subject Six smoked a cigarette he produced: a large increase in 12-20 Hz activity (Panel 3, Phase B); an increase in heart rate (Panel 5, Phase B); a moderate decrease in 8-12 Hz activity (Panel 1, Phase B); a decrease in muscle tension (Panel 4, Phase B); and, a decrease in skin temperature (Panel 6, Phase B).

Within a 20 minute time period after smoking a cigarette Subject Six produced: (1) an initial moderate increase in 12-20 activity, which recovered towards baseline levels by the end of the session (Panel 3, Phase B); (2) an initial increase in heart rate, which also recovered towards baseline (Panel 5, Phase B); (3) a moderate decrease in 8-12 Hz activity, which recovered towards baseline levels (Panel 1, Phase B): and, (4) a decrease in skin temperature, which remained below baseline levels throughout the session (Panel 6, Phase B). There also appeared to be an initial increase in frequency of 8-12 Hz activity which recovered towards baseline approximately ten minutes after he had smoked a cigarette.

During the 8-12 Hz Feedback Phase of the experiment Subject Six moderately increased his 8-12 Hz activity (Panel 1, Phase C). In conjunction with the increase of his 8-12 Hz activity Subject Six's

temperature increased (Panel 6, Phase C) and his frequency of 8-12 Hz activity decreased slightly (Panel 8, Phase C). Muscle tension possibly decreased, however, since the mean of Baseline (A2) did not return to the mean Baseline (A1) level, and because of the extreme variability in the Baseline (A2) Phase the author is cautious in making this interpretation (Panel 4, Phase C). The amplitude of 8-12 Hz activity mean data also demonstrated a lack of return of Baseline (A2) to the mean level of Baseline (A1) (Panel 7, Phase C).

After completing the 8-12 Hz occipital EEG training Fadeout Phase Subject Six's: 4-8 Hz activity increased largely as compared to Baseline (A1) and (A2) Phases (Panel 2, Phase D); skin temperature increased (Panel 6, Phase D), but was slightly lower than in the training phase; 12-20 Hz activity decreased moderately (Panel 3, Phase D); muscle tension decreased (Panel 4, Phase D); heart rate decreased (Panel 5, Phase D); and, frequency of 8-12 Hz activity decreased (Panel 1, Phase D). Both muscle tension, which has been discussed above, (Panel 4, Phase D) and the amplitude of 8-12 Hz activity (Panel 7, Phase D), have mean Baseline (A2) levels which did not return to Baseline (A1) levels so interpretation of these data is difficult.

It is important to note that when Subject Six was proceeding through the Fadeout Phase he produced an increase in his 4-8 Hz activity. In conjunction with his 4-8 Hz activity increasing largely he also produced a moderate 12-20 Hz decrease, a muscle tension decrease and a heart rate decrease. His skin temperature increased and his frequency of 8-12 Hz activity decreased. None of these physiological parameters changed in the same direction when he smoked, with the possible exception of a decrease in his muscle tension. It should also be noted that Subject

Six reported to the author that during the 6-month follow-up period he and his wife relocated their living quarters with relatives, where smoking was not permitted in the house. Subject Six stated that he believed this accounted for some decrease in his cigarette smoking frequency. Subject Six's smoking frequency data indicated that he decreased his smoking frequency 41%, from 17 to 10 cigarettes per day.

An overall summary of the data indicates the following general findings for each subject:

Subject One's average smoking rate was 17 cigarettes per day at the beginning of the study. His 8-12 Hz activity increased moderately as a result of smoking, indicating that his brain waves were slowing down. At the completion of the study Subject One showed that he could increase his 8-12 Hz activity largely without biofeedback. In conjunction with his 8-12 Hz activity alteration, his heart rate and 12-20 Hz activity changed in the same direction at the completion of the study as it did as a result of smoking a single cigarette. Subject One quit smoking.

Subject Two's average number of cigarettes smoked per day was 38 at the beginning of the study. His 4-8 Hz activity increased moderately as a result of smoking, thus his brain waves slowed down. At the completion of the Fadeout Phase, Subject Two was successful in increasing his 8-12 Hz activity moderately. As he increased his 8-12 Hz activity moderately he also decreased his 12-20 Hz activity slightly, increased his heart rate, and increased his skin temperature. These physiological parameters changed in the same direction as a result of Subject Two's smoking a single cigarette. Subject Two had quit smoking at the end of the follow-up period.

Subject Three's average daily cigarette frequency was 38. As a result of smoking a single cigarette his 4-8 Hz activity increased slightly, which suggests a slowing down of the brain wave activity. During the Fadeout Phase of the experiment Subject Three could not increase his 8-12 Hz activity. However, he was able to increase his heart rate, his skin temperature, his frequency of 8-12 Hz activity and possibly his amplitude of 8-12 Hz activity when he was provided with 8-12 Hz feedback. Subject Three had not quit smoking, but he has decreased smoking to 15 cigarettes per day, a 61% decrease.

Subject Four's average number of cigarettes smoked per day was 50 at the beginning of the study. As a result of smoking one cigarette his 4-8 Hz activity increased slightly which possibly suggests a slowing down of his brain waves (Note the phase indicates a lack of return to baseline A1 levels). At the completion of the study Subject Four could not increase his 8-12 Hz activity without the use of the feedback. In conjunction with the instructions to increase his 8-12 Hz activity he did, however, increase his 4-8 Hz activity largely and his heart rate, which also occurred as a result of his smoking a single cigarette. Subject Four had not quit smoking at the time of the follow-up period and had only decreased 12% from his baseline smoking rate.

Subject Five's average number of cigarettes smoked per day was 18. As a result of smoking his brain wave pattern initially slowed down, but then speeded up demonstrating a slight increase in 8-12 Hz activity. At the completion of the Fadeout Phase it was concluded that Subject Five could not increase his 8-12 Hz activity without the use of the 8-12 Hz biofeedback signal. In conjunction with Subject Five's

attempts to increase his 8-12 Hz activity the data indicated that his heart rate increased and his amplitude of 8-12 Hz activity increased. These two parameters changed in the same direction as when he smoked a single cigarette. Subject Five had not quit smoking, but did decrease his smoking frequency by approximately 17% from baseline levels.

Subject Six's average number of cigarettes smoked per day was 17 at the beginning of the experiment. As a result of his smoking one cigarette, his 12-20 Hz activity increased largely which represented a speeding up of his brain waves. At the conclusion of the Fadeout Phase of the study Subject Six was unable to increase his 8-12 Hz activity above baseline levels. However, during the Fadeout Phase there did appear to be a decrease in his muscle tension. This decrease also appeared during and immediately after his smoking of a cigarette. By the end of the follow-up period Subject Six had not quit smoking, but did decrease his smoking frequency by about 41% from baseline levels.

General Discussion

Stephens (1977), in his review of physiological variables in cigarette smoking, has stated that, "Little effort has been directed toward identification of the physiological parameters and individual physiological differences associated with smoking". The identification of the individual physiological differences associated with smoking cigarettes is valuable because it could contribute to the development of more successful cessation treatment programs, and it could provide information for why most cessation treatment programs are successful with only a certain subgroup of the total population of cigarette smokers

who wish to quit smoking. In addition to the lack of studies which have investigated the effects of smoking across an individual's physiological parameters, there also appear to be only three studies (Havelick, 1977; Kothare, 1975; Turin & Nideffer, 1974) which have attempted to decrease smoking frequency via any facsimile of a biofeedback approach. Hence, the importance of this study is due, in a large part, to its pioneering nature in the field of smoking reduction and biofeedback.

The purpose of the present study was two-fold: 1) to objectively document the immediate effects and after effects that smoking a cigarette has on an individual's physiology (especially, the effect that smoking of a cigarette has on a smoker's brain wave pattern, muscle tension, heart rate and skin temperature), and 2) to explore the usefulness of a new physiological smoking cessation treatment technique, whereby the cigarette smoker is trained to increase his 8-12 Hz occipital EEG activity level via a biofeedback procedure whenever he has the urge to smoke. It is suggested that his type of procedure will provide the smoker with a self-induced physiological substitute for smoking rather than a temporary smoking-induced physiological change, which he acquired after he smokes a cigarette.

The immediate and after-effects of smoking a cigarette on physiology and the effect of 8-12 Hz occipital EEG training on the frequency of cigarette smoking was determined via the use of a multiple baseline across subjects design. The use of this design in this study required that each subject proceeded through a series of five phase conditions staggered across time: Baseline (A1), Smoking (B), Baseline (A2), Feedback (C), and Fadeout (D). In multiple baseline terminology

this design is referred to as an ABACD design. Thus, purpose number one (as presented above) is researched by the use of multiple baseline reversal design ABA and purpose number two is researched by the use of a AC design across subjects.

The results of the present study indicated that the majority of the smokers produced increases in 4-8 Hz activity and increases in heart rate while they were smoking a cigarette. Immediately after the smoking of a cigarette (defined in the present study as occurring within a 20-minute time period after the cigarette has been extinguished) the majority of the smokers demonstrated increases in their heart rates and decreases in their skin temperatures. Heart rate increases as a result of smoking which were found in this study, coincide with the findings of around 90 other publications (Stephens, 1977). Skin temperature decreases have also been documented in a number of studies some of which are: Auge, 1973; Frankenhauser, Myrsten, Waszak, Neri & Post, 1968; Larson, Haag, & Silvette, 1961. Although the present data indicated that there were no consistent brain wave pattern changes across the subjects immediately after the smoking of a cigarette, there were increases in: 4-8 Hz activity for three subjects; 8-12 Hz activity for two subjects; and, 12-20 Hz activity for one subject. A closer analysis of the individual subject data indicated that four of the smoker's brain wave patterns shifted from producing a higher percentage of faster brain waves to that of producing a higher percentage of slower brain waves after they had smoked a single cigarette. This suggests a slowing down of the brain wave pattern for these four subjects. The other two subjects data suggested a speeding up of their brain wave

pattern after the smoking of a cigarette. More specifically for the two subjects who demonstrated a speeding up of their brain waves, Subject Five demonstrated an increase in 8-12 Hz activity, while his 4-8 Hz activity decreased and his 12-20 Hz activity remained relatively unchanged, which suggests a speeding up of 8-12 Hz activity; and, (2) Subject Six demonstrated an increase in 12-20 Hz activity with a decrease in both his 4-8 Hz and 8-12 Hz activity, which suggest a speeding up of 12-20 Hz activity. These data aid in clarifying the controversy of whether the smoking of a cigarette speeds up or slows down brain wave activity. Brown (1968); Itil, Ulett, Hsu, Klingenberg, and Ulett (1971); and Phillips (1971), suggests that the smoking of a cigarette produces a tranquilizing effect, or a general slowing down of the brain wave activity. Contrary to Brown's hypothesis Lambiase and Serra (1957); Hauser, Schwartz, Roth and Bickford (1958); Bickford (1960); Weschsler (1962); Murphree, Pheifer and Price (1967); Murphree and Schultz (1968); Phillips (1971) and others suggest that smoking acts as a stimulant and speeds up brain wave activity. The data gathered in the present study suggested that the speeding up or slowing down of the brain wave activity is individual specific and that, in fact, some smokers (for example Subject Five) demonstrated an initial slowing down of the brain wave activity while actually smoking, but a reversal to a speeding up pattern shortly after the cigarette had been smoked.

During the 8-12 Hz occipital EEG training Feedback Phase of the experiment, four of the six subjects demonstrated that they had acquired the ability to increase their 8-12 Hz activity when they were

provided music feedback for their 8-12 Hz activity production. However, during the 8-12 Hz occipital EEG training Fadeout Phase only two of these four smokers were able to continue producing high levels of 8-12 Hz activity without the benefit of the biofeedback signal. These two smokers have quit smoking cigarettes completely at the end of a six-month follow-up period and, when contacted by phone at the eight-month follow-up period, they reported that they were still abstaining from any cigarette smoking. The other subjects decreased 61%, 41%, 17%, and 12% from their original average smoking frequency at the beginning of the study. The possible reasons for why these four subjects decreased their smoking frequency, but had not learned to control their 8-12 Hz activity is discussed in the following sections.

As a result of the findings of this study there appears to be two major issues which warrant some discussion. First, the results of the present study are more detailed than the findings of earlier studies, concerning the effects smoking a cigarette has on EEG patterns. For example, Brown (1974) Itil et al., (1971), Phillips (1971), and others have suggested a general slowing down of an individual's brain waves after smoking a cigarette, while Murphree et al., (1967), Ulett and Itil (1969) and others have suggested a general speeding up of one's brain waves after smoking. The results of the present study indicate that 5 out of 6 smokers in this study (S1, S2, S3, S4, S5) produced an increase in 4-8 Hz brain waves while actually smoking a cigarette. The immediate after effect of smoking a cigarette was a continued production of 4-8 Hz activity for S2, S3, and S4. However, for S1 and S5 there was an increase in 8-12 Hz activity and for S6 there was no

parameters, indicates that smoker's physiologies are affected differently as a result of smoking. Perhaps one key reason why our present-day-smoking-treatment therapies have been generally ineffective is because the cigarette produces different physiological changes for different subjects and, thus, one treatment technique is not successful for all smokers. For example, if a person smokes a cigarette and his brain wave patterns speeds up, his muscle tension increases, and his skin temperature decreases he probably won't respond to a treatment technique which assumes that he smokes to relax and thus, provides him with relaxation therapy. In contrast this type of smoker would possibly decrease his smoking frequency more significantly when he was taught a procedure to stimulate his physiology. It is deemed important by this author that smoking researchers and therapists begin to look more closely at what smoking produces physiologically for the smoker. To date psychologists have tended to focus on the behavioral aspects and ignore the physiological aspects.

Concerning the usefulness of the new biofeedback treatment procedures utilized in the present study, there are two basic issues which the author suggests must be dealt with: 1) why did two subjects quit smoking, and 2) why did the other subjects decrease their smoking frequency to some extent? One hypothesis is that if the brain wave pattern alterations that are produced by cigarette smoking are re-produced via biofeedback procedures, then the smoker will quit smoking. This is especially evident from Subject One's data. Subject One produced a clear increase in 8-12 Hz activity after the smoking of a cigarette and when he was provided EEG training procedures, which

taught him how to increase his 8-12 Hz activity without the smoking of a cigarette; he quit smoking. Subject One is perhaps the best demonstration of what the author believes to be the appropriate use of biofeedback training to provide a smoker with a method of substituting his self-induced brain wave activity alteration for a smoking-induced alteration in brain wave activity.

Subject Two's data present a slight deviation from the first hypothesis suggested above. His data indicate that he produced an increase in 4-8 Hz activity as a result of smoking, and this fact along with a decrease of 12-20 Hz activity and 8-12 Hz activity, suggests a general slowing down of his brain waves. In this case the 8-12 Hz activity training provided him with a method of slowing down his brain waves (e.g., decreasing 12-20 Hz activity) which as the data indicates, was the same effect he got when he smoked a cigarette. Thus, Subject Two quit smoking because he was provided with a method of slowing down his brain waves rather than duplicating the exact brain wave pattern produced by smoking.

Another theory, which is suggested by the data, is that possibly it is not the particular brain wave pattern variation that is important, but rather the overall number of physiological parameters that change while smoking which coincide with physiological parameters taught to be altered via biofeedback training. For example, both Subjects One and Two had three physiological parameters which changed in the same direction during training as they did when they smoked a cigarette. Subject One produced increases in 8-12 Hz activity, increases in heart rate, and decreases in 12-20 Hz activity all of which changed in the

only work for subjects who produced less 12-20 Hz activity as a result of smoking and not merely a general slowing down of the brain waves. A fifth and final hypothesis is that none of these hypotheses are relevant and that the smokers lied to the experimentor about their smoking frequency. Since there were no reliability checks made on smoking frequency, the author can not be positive that the smokers were truthful about their smoking frequency.

In summary, the five possible hypothesis for the smokers quitting smoking as a result of biofeedback training are:

1) A smoker smokes to produce a particular brain wave pattern. If this brain wave pattern can be duplicated via biofeedback procedure the smoker will quit smoking cigarettes;

2) A smoker smokes to produce a general slowing down or speeding up of his brain wave pattern. If a general slowing down or speeding up pattern of the brain waves can be duplicated via biofeedback training procedure the smoker will reduce smoking cigarettes;

3) A smoker smokes to produce particular alterations in one or more different physiological parameters such as muscle tension, heart rate, skin temperature, etc. If these particular physiological parameters, or more than two of them, can be duplicated via biofeedback procedure the smoker will quit smoking cigarettes;

4) Only smokers who smoke to reduce 12-20 Hz brain waves from their EEG will reduce smoking as a result of 8-12 Hz biofeedback training; and,

5) The smokers did not accurately report their smoking frequency.

In conclusion some possible treatment procedures which need further exploration as to their usefulness are suggested. One treatment procedure which needs to be tested is to train the smoker to produce the exact brain wave pattern change that a cigarette produces, such as was done for Subject One. Another treatment procedure which needs to be tested would focus on not only the smoker's brain wave pattern, but also on training the subject to alter all of those physiological parameters which move in a particular direction as a result of smoking. For example, if skin temperature decreased and heart rate increased and 4-8 Hz activity increased as a result of smoking for one subject then that subject should be trained to alter all of those physiological parameters in the same direction as they proceed when he smoked.

It should be noted that if all of these physiological treatment approaches for decreasing cigarette smoking turn out to be ineffective treatment procedures in and of themselves, their use in conjunction with some other behavior treatment approaches could be explored. There is also the possibility that the physiological monitoring could be utilized as an evaluation technique to determine whether a subject will respond to a particular type of treatment. For example, if a subject reports that he smokes to relax and a therapist decided to provide the subject with relaxation therapy, the therapist should first monitor the subject smoker's physiology to determine if the subject's muscle tension decreases during smoking and if his EEG suggests a slowing down pattern as a result of smoking.

One additional suggestion for future research is that post checks be taken on the smoker's abilities to control certain physiological parameters over a substantial time period (3 months or more) following the Training and Fadeout Phases. The post check would aid in assuring the investigator that the ability to control ones brain waves was consistent across an extended time period. The present investigator attempted to post check his smokers, however, only one subject remained within area to allow for such testing. This smoker had unfortunately not been one of the smokers who had quit smoking, thus his post check only confirmed the previous fadeout data which indicated that he did not acquire the ability to increase his 8-12 Hz activity without the biofeedback signal. As is true in most research studies, there are limitations. The following limitations of this study are listed. By no means are these the only limitations, however, these are the ones the author sees as limitations which need to be controlled for in further research studies:

1) only motivated subjects were used in this study;
2) only male subjects were used;
3) only one type of music was utilized as feedback;
4) there was no reliability checks on frequency of cigarette smoking;
5) the experiment's observers possibly could have inflated their reliability score, by monitoring each others recordings; and,
6) the degree of change of physiological parameters, which is needed to produce a change in overt behavior is unknown

at the present time and thus the degree of change in this study is possibly insignificant.

In conclusion the present study investigated the usefulness of a biofeedback training approach to decrease in frequency of cigarette smoking. For two of the subjects the procedures proved effective, however, there is presently not enough research conducted in this area to conclude that the technique is of value. The author made some suggestions for further research, which could aid in investigating the usefulness of a biofeedback approach to decrease cigarette smoking frequency.

Table 2

Average Number of Cigarettes Smoked Per Day

Subject and type of Smoker	Base-line A_1	Smoking B	Base-line A_2	Feed-back C	Fade-out D	3-Mo. Follow-up	6-Mo. Follow-up	Percent Dec. From A_1 to 6-Mo. Follow-up
S-1 (moderate)	17	18	11	5	0	0	0	100%
S-2 (heavy)	38	34	53	24	11	0	0	100%
S-3 (heavy)	38	37	29	24	13	11	15	61%
S-4 (heavy)	50	48	49	44	46	43	44	12%
S-5 (moderate)	18	20	18	15	14	6	15	17%
S-6 (moderate)	17	16	17	16	14	11	10	41%

Figure Legend

In the following figures (5-18) the data points shown represent the means of five minute segments across the total number of sessions conducted during the first three phases (Baseline A1, Smoking B, and Baseline A2). For the remaining two phases (Feedback C and Fadeout D) the data points shown also represent the means of five-minute segments, however, the means are calculated from only the last three sessions for each phase. The shaded area represents variability of these data points (standard deviation). The broken horizontal lines represent the means of the data points during that phase. The first data point in each phase represents a period in which physiological recordings were taken in the absence of any further treatment. During the Smoking (B) phase note that during the second 5-minute period (see data point marked "X") the subjects smoked a single cigarette.

Figure 5. Mean percent time alpha production (8-12 Hz) per subject for each phase condition.

FIGURE 5

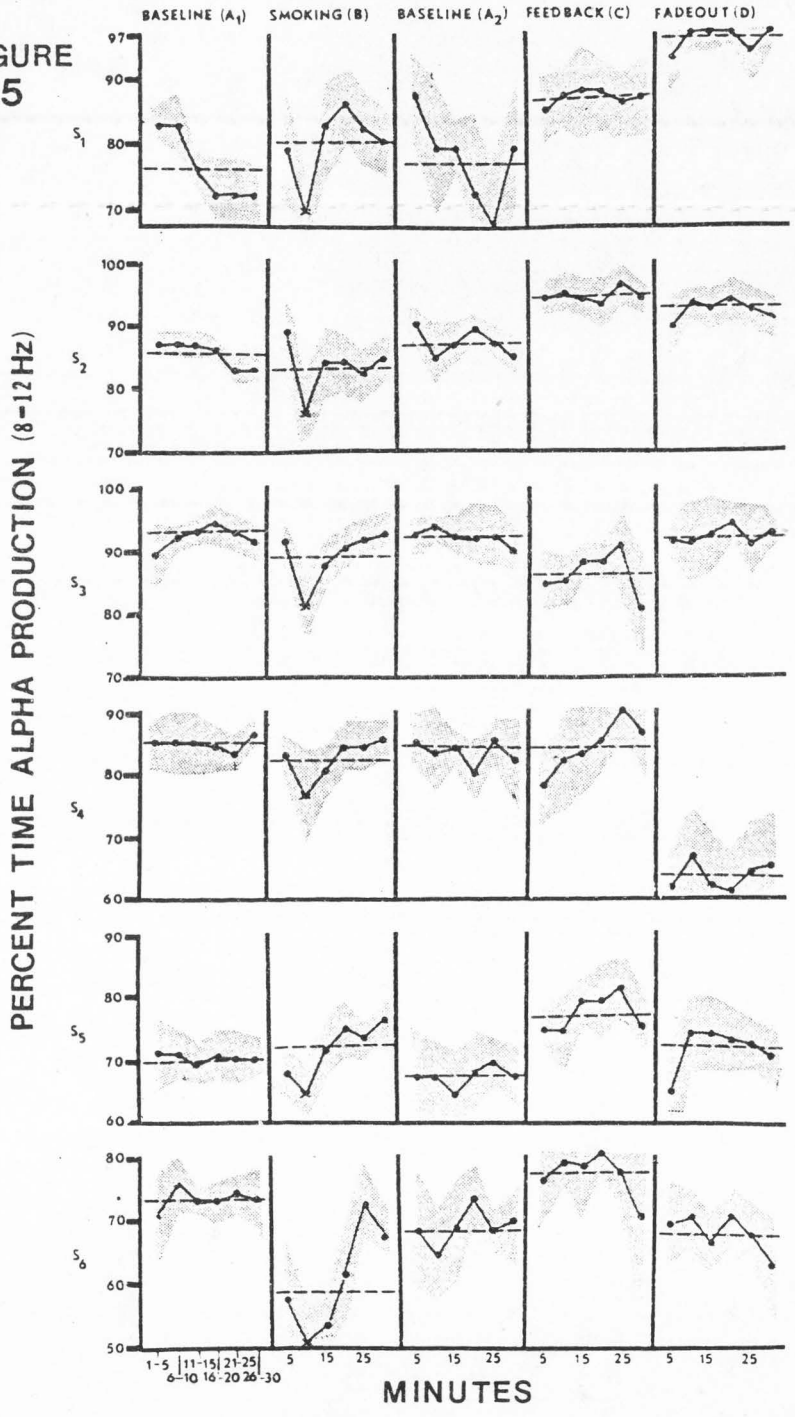

BASELINE (A1)
SMOKING (B)
BASELINE (A2)
FEEDBACK (C)
FADEOUT (D)
PERCENT TIME ALPHA PRODUCTION (8-12Hz)
S1
S2
S3
S4
S5
S6
1-5
6-10
11-15
16-20
21-25
26-30
MINUTES

Figure 6. Mean amplitude of alpha (8-12 Hz) per subject for each phase condition.

FIGURE 6

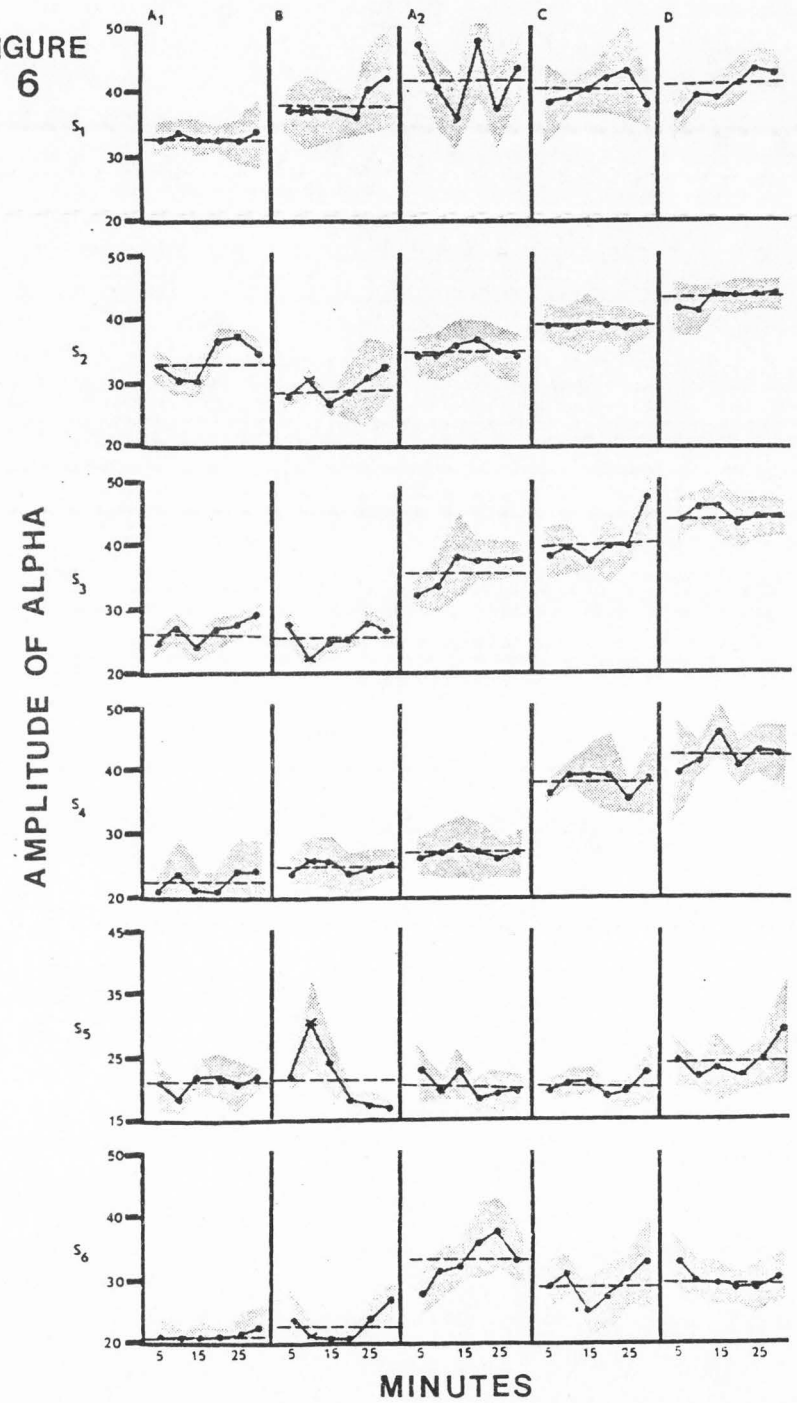


Figure 7. Mean frequency of alpha (8-12 Hz) per subject for each phase condition.

FIGURE 7

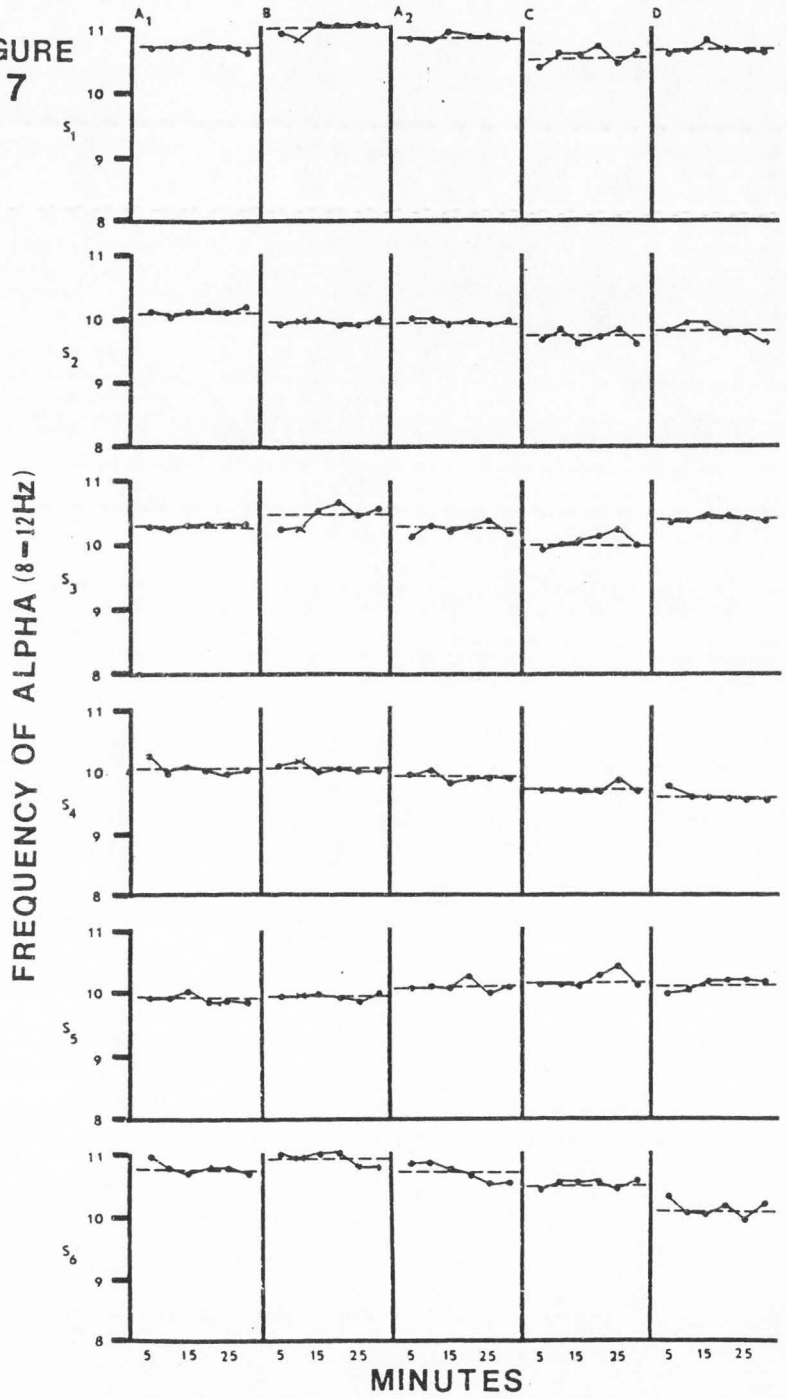

A1
B
A2
C
D
S1
S2
S3
S4
S5
S6
11
10
9
8
FREQUENCY OF ALPHA (8-12Hz)
5 15 25 5 15 25 5 15 25 5 15 25 5 15 25
MINUTES

Figure 8. Mean percent time theta production (4-8 Hz) per subject for each phase condition.

FIGURE 8

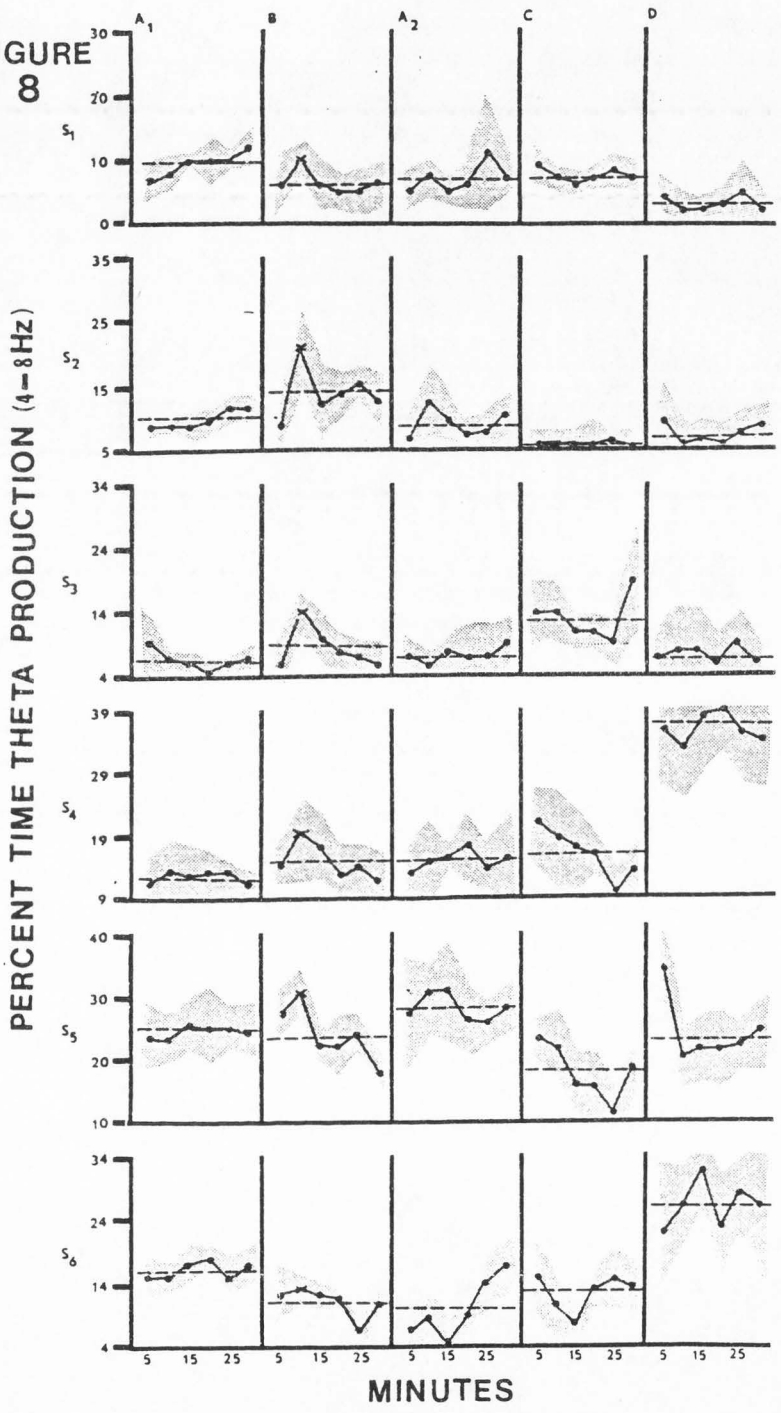

A1
B
A2
C
D
S1
S2
S3
S4
S5
S6
PERCENT TIME THETA PRODUCTION (4-8Hz)
MINUTES
5
15
25

Figure 9. Mean percent time beta production (12-20 Hz) per subject for each phase condition.

FIGURE 9

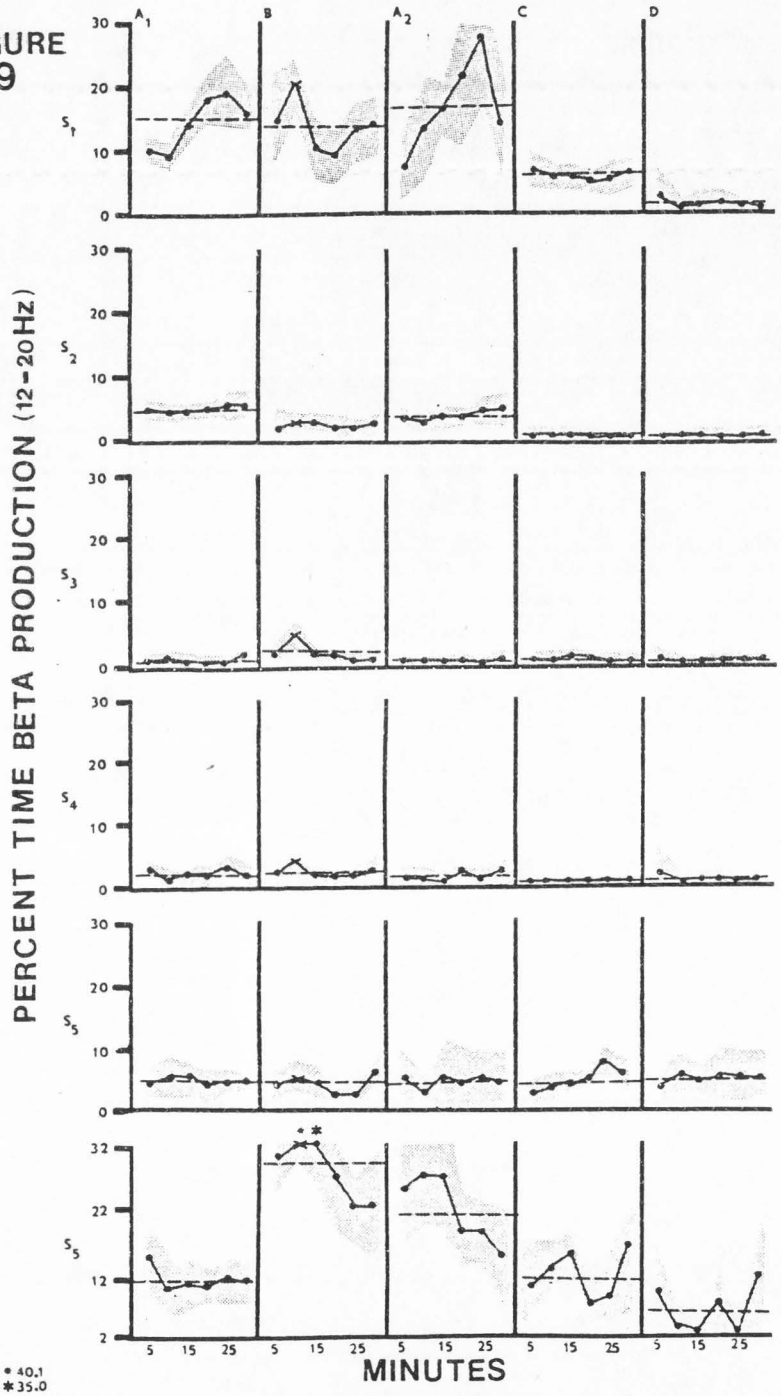

A1
B
A2
C
D
S1
S2
S3
S4
S5
S5
PERCENT TIME BETA PRODUCTION (12-20Hz)
MINUTES
5 15 25
* 40.1
* 35.0

Figure 10. Mean microvolts of muscle tension per subject for each phase condition.

FIGURE 10

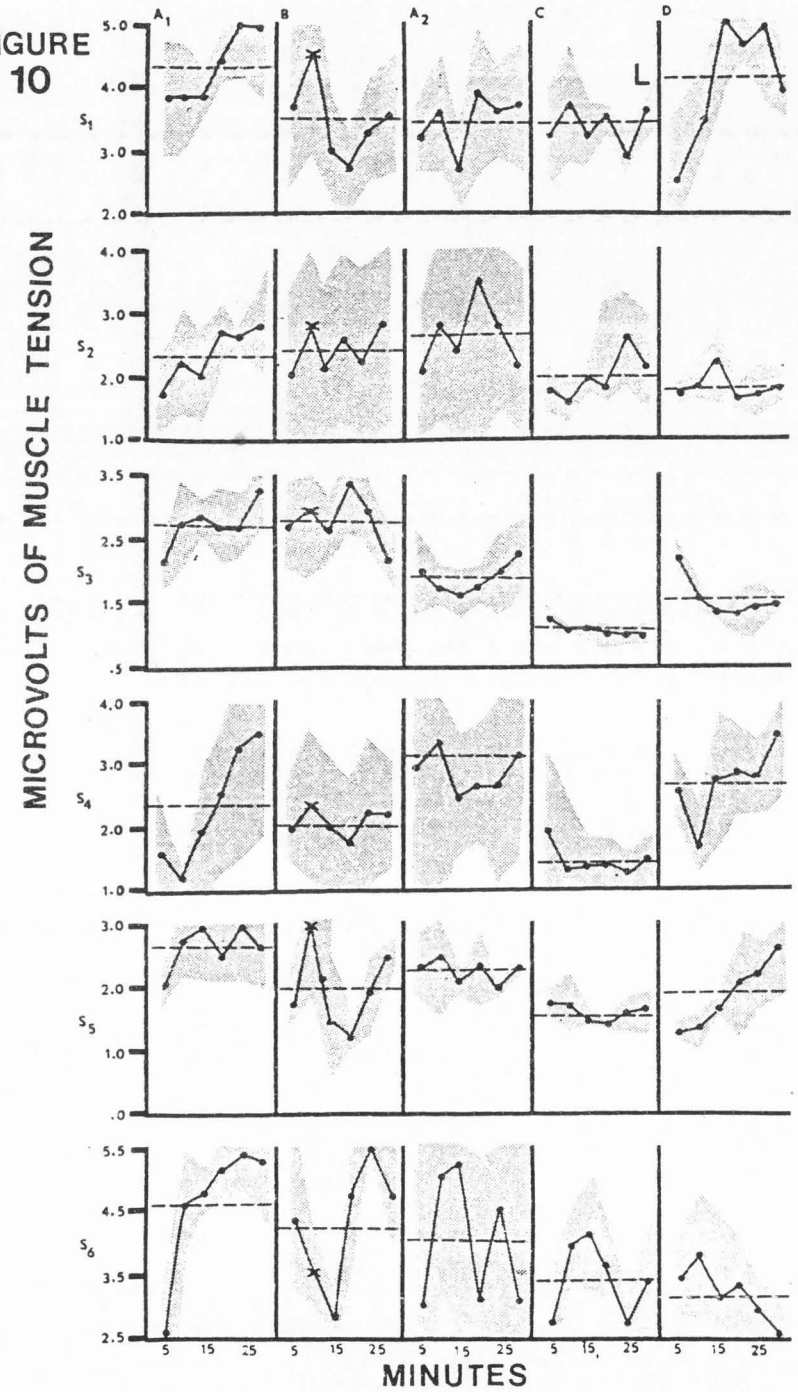

A1
B
A2
C
D
L
MICROVOLTS OF MUSCLE TENSION
S1
S2
S3
S4
S5
S6
MINUTES
5
15
25

Figure 11. Mean heart beats per minute per subject for each phase condition.

FIGURE 11

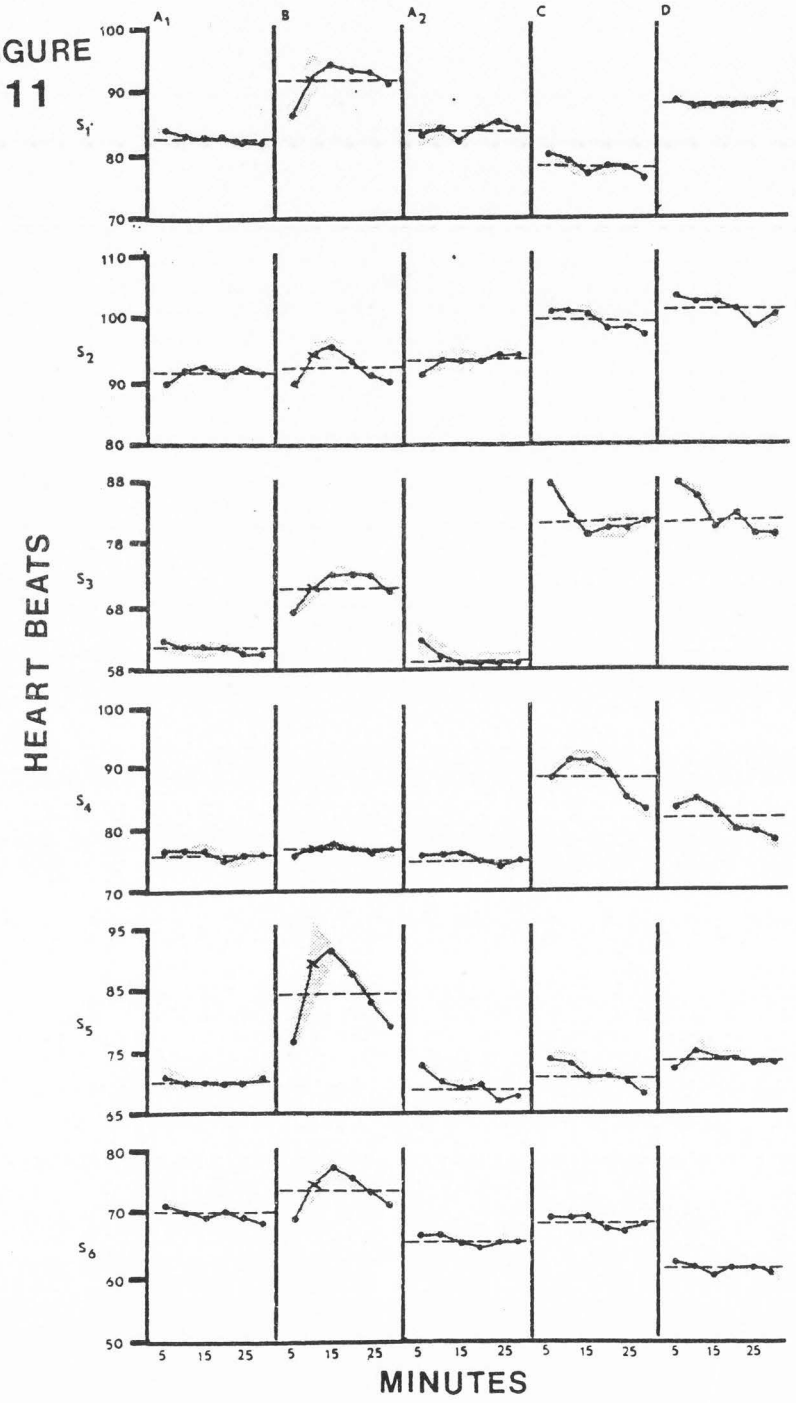


Figure 12. Mean degrees of skin temperature on subject for each phase condition.

FIGURE 12

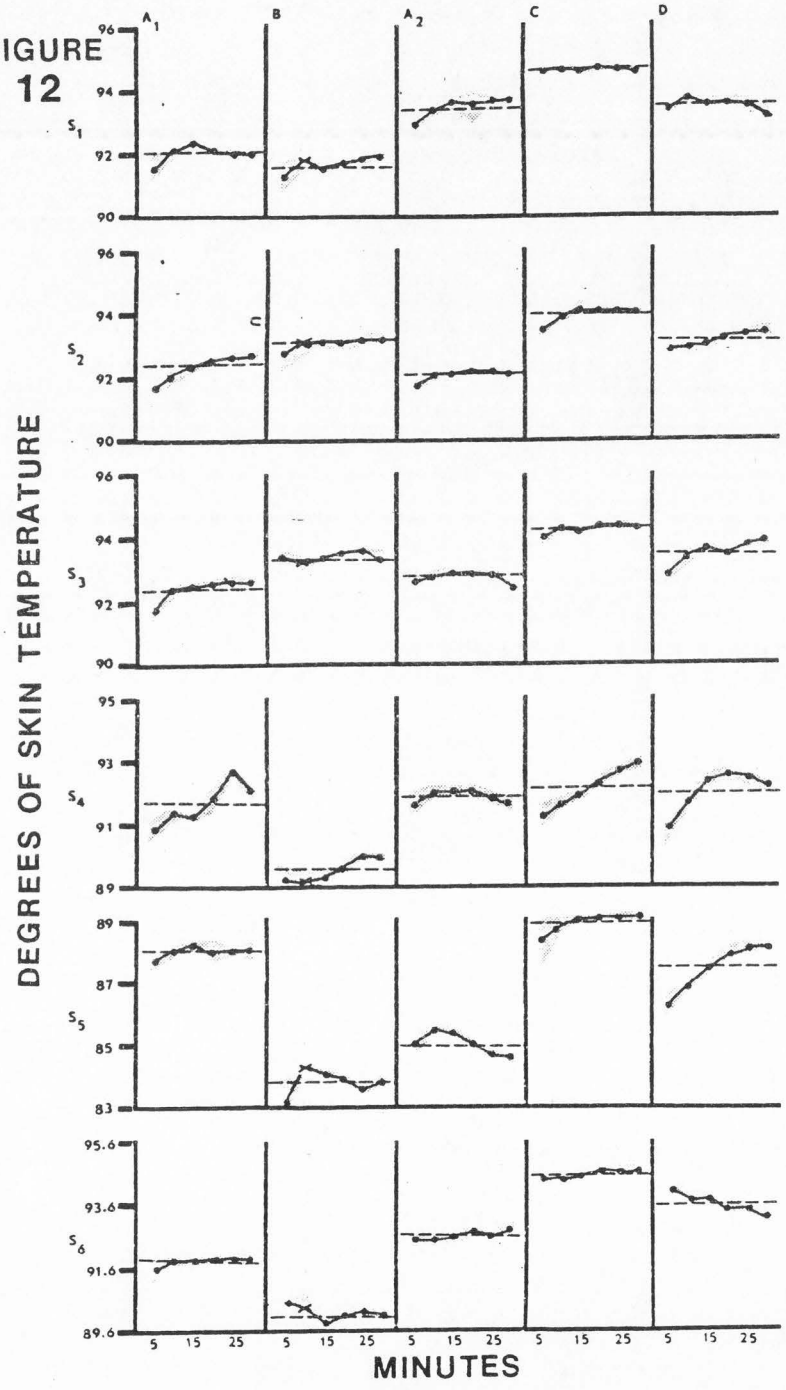

A1
B
A2
C
D
S1
S2
S3
S4
S5
S6
96
94
92
90
95
93
91
89
87
85
83
95.6
93.6
91.6
89.6
5 15 25
DEGREES OF SKIN TEMPERATURE
MINUTES

Figure 13. Data for Subject One.

FIGURE
13

SUBJECT
1

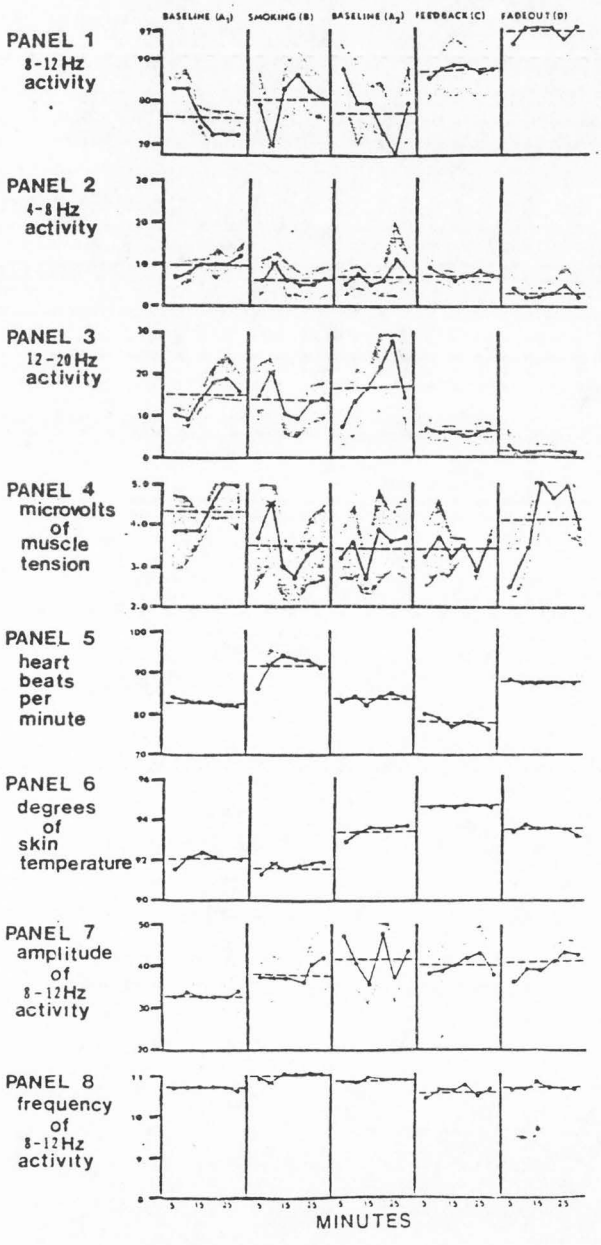


Figure 14. Data for Subject Two.

FIGURE
14

SUBJECT
2

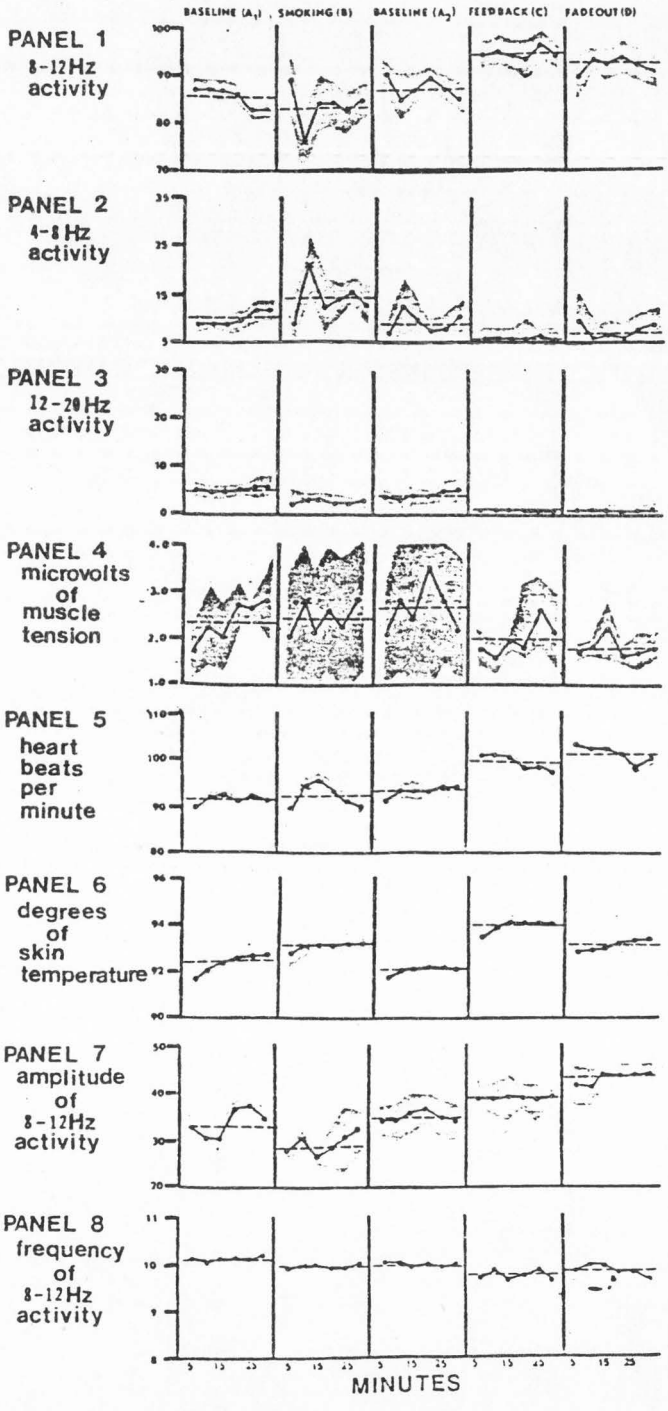


Figure 15. Data for Subject Three.

FIGURE 15

SUBJECT 3

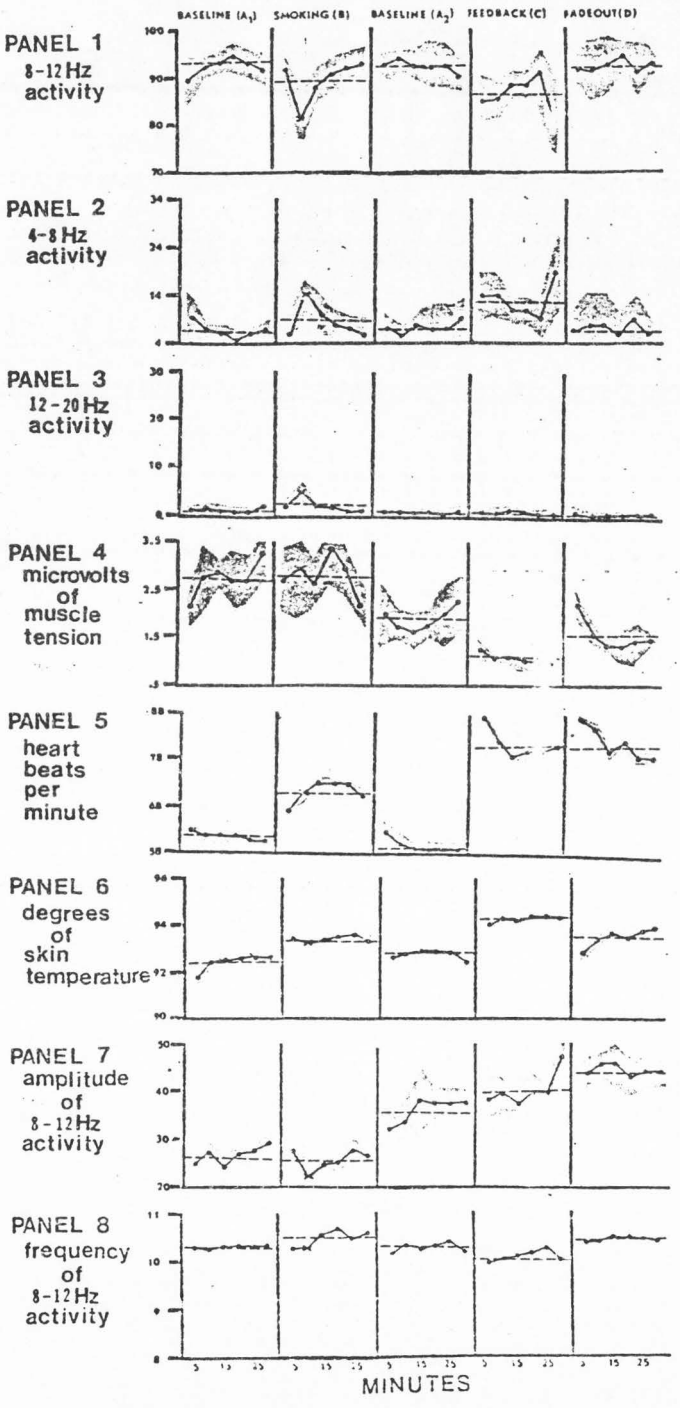


Figure 16. Data for Subject Four.

FIGURE 16

SUBJECT 4

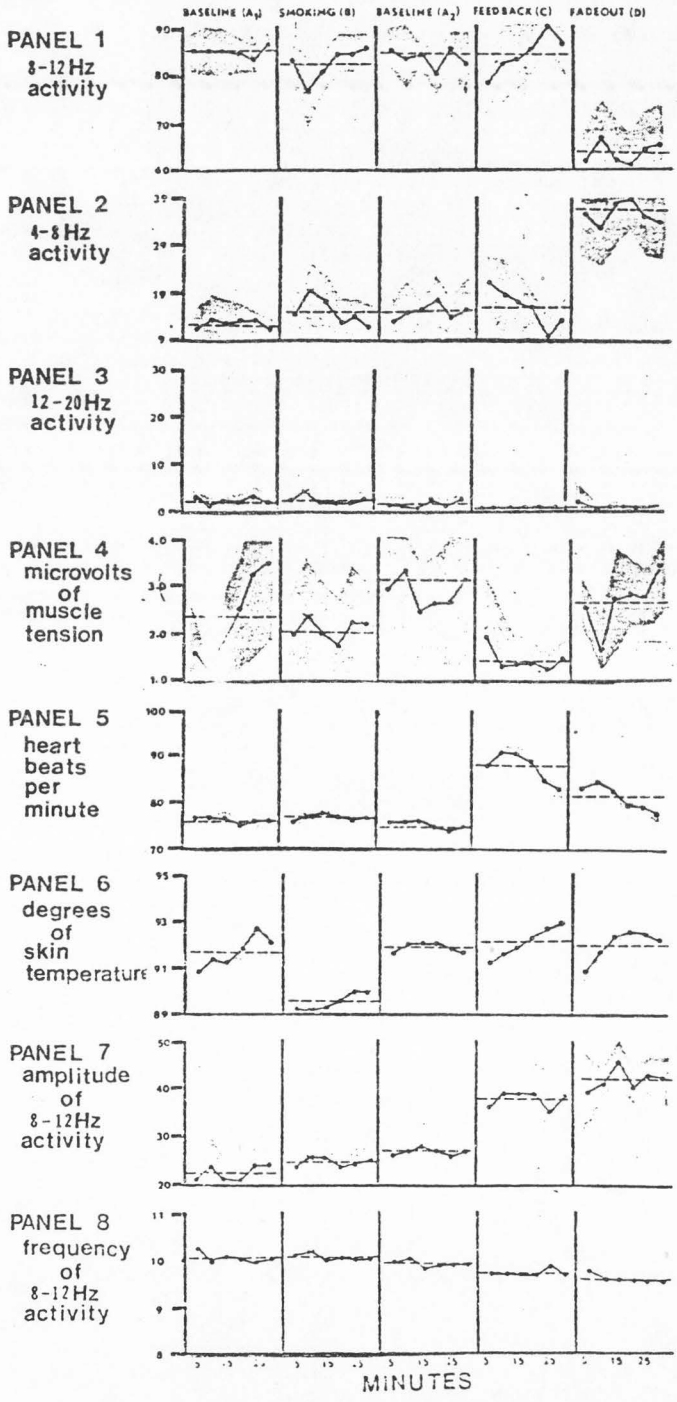


Figure 17. Data for Subject Five.

FIGURE 17

SUBJECT 5

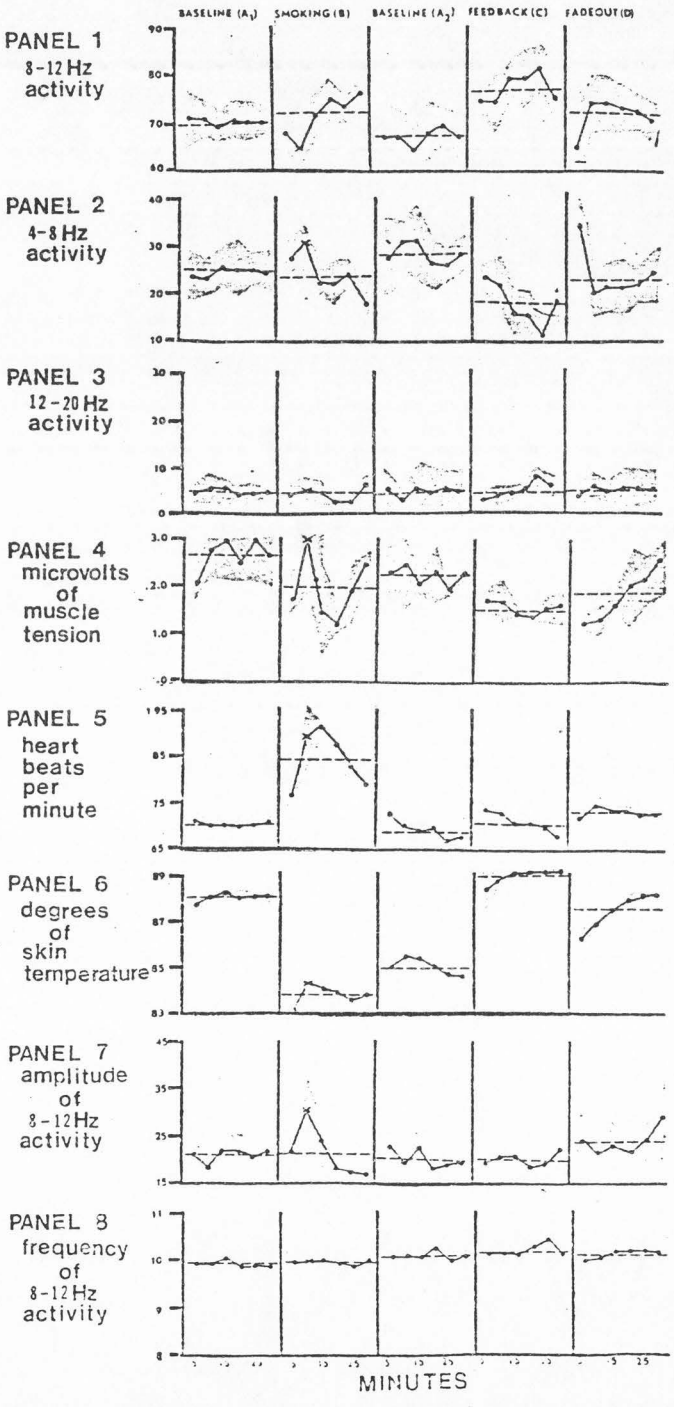
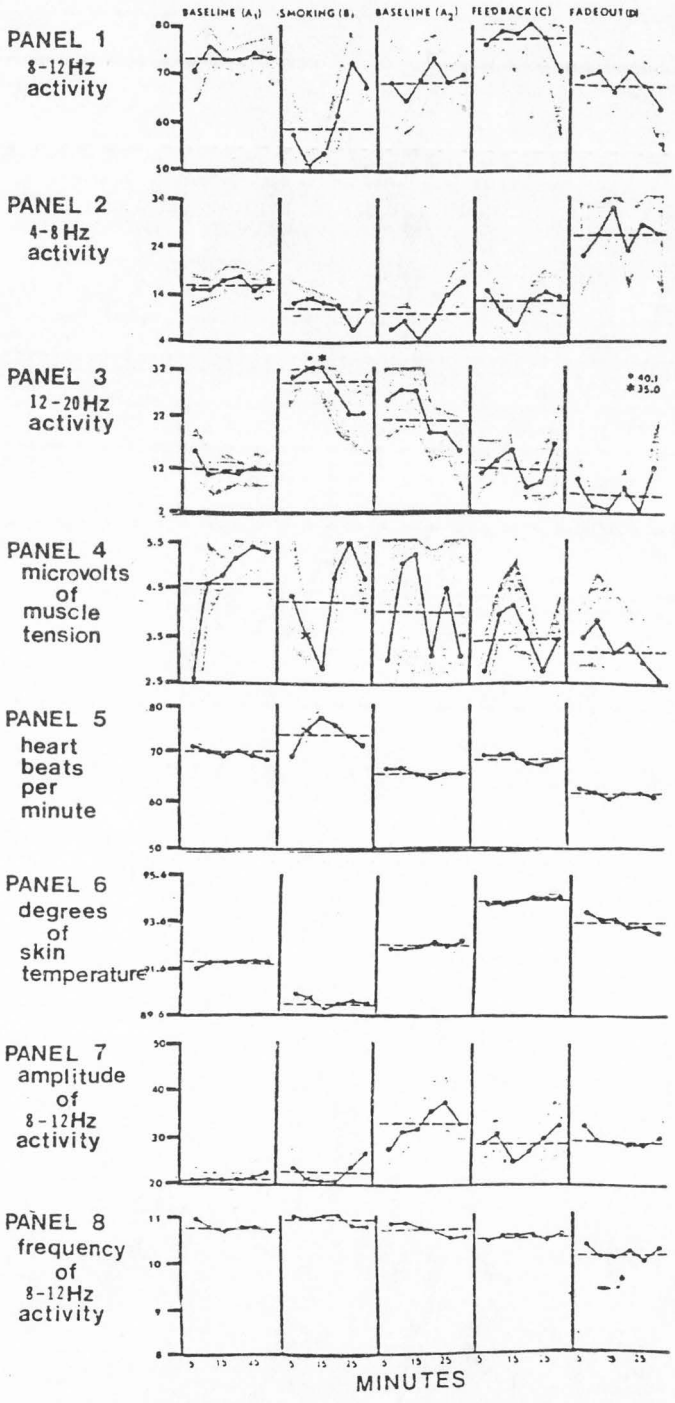


Figure 18. Data for Subject Six.

FIGURE 18

SUBJECT 6



REFERENCES

Armitage, A. K., Hall, G. H., & Morrison, C. F. Pharmacological basis for the tobacco smoking habit. Nature (London), 1968, 217, 331-334.

Auge, C. Smoking patterns, nicotine intake at different times of day and changes in two cardiovascular variables while smoking cigarettes. Psychopharmacologia, 1973, 30, 135-144.

Auger, T. J., Wright, E., & Simpson, R. H. Posters as smoking deterrents. Journal of Applied Psychology, 1972, 52, 169-171.

Baer, D. M., Wolf, M. M., & Risley, T. E. Some current dimensions of applied behavior analysis. Journal of Applied Behavior Analysis, 1968, 1, 91-97.

Bandura, A. Principles of behavior modification. New York: Holt, Rinehart, & Winston, 1969.

Barnes, T. C. Cerebral effects of cigarette smoke. Paper presented at the American Society of Pharmacology and Experimental Therapeutic Meeting, Mexico City, July 1966. The Pharmacologist, 1966, 8, 219.

Bartlett, W. A., & Whitehead, R. W. The effectiveness of meprobamate and lobeline as smoking deterrents. Journal of Laboratory and Clinical Medicine, 1957, 50, 278-281.

Beatty, J. Similar effects of feedback signals and instructional information on EEG activity. Physiology and Behavior, 1972, 9, 151-154.

Berglund, E., Bernstein, O. A., Eisinger, R. A., Hochbaum, G. M., Lichtenstein, E., Schwartz, J. L., & Straits, B. C. Guidelines for research on the effectiveness of smoking cessation programs, a committee report. (Received from the National Interagency Council on Smoking and Health, 419 Park Avenue, South, New York City, New York 10016.)

Bernard, H. S., & Efran, J. S. Eliminating versus reducing smoking using pocket timers. Behavior Research and Therapy, 1972, 10, 399-401.

Bernstein, D. A. Modification of smoking behavior: An evaluative review. Psychological Bulletin, 1969, 71, 418-440.

Bernstein, D. A., & McAlister, A. The modification of smoking behavior: Progress and problems. Addictive Behaviors, 1976, 1, 89-102.

Best, J. A., & Steffy, R. A. Smoking modification procedures tailored to subject characteristics. Behavior Therapy, 1971, 2, 171-191.

Bickford, R. Physiology and drug action: An electroencephalographic analysis. Federation Proceedings, 1960, 19, 619-625.

Black, A. H. The operant conditioning of central nervous system electrical activity. In G. H. Bower (Ed.), The Psychology of Learning and Motivation. New York: Academic Press, 1972, 6.

Bovet, D., Bovet-Nitti, F., & Oliverio, A. Effects of nicotine of avoidance conditioning in inbred strains of mice. Psychopharmacologue, 1966, 10, 1-5.

Bovet, D., Bovet-Nitti, F., & Oliverio, A. Action of nicotine on spontaneous and acquired behaviors in rats and mice: The effects of nicotine and smoking on the central nervous system. Academic Science, 1967, 142, 261-267.

Brown, B. B. Some characteristics EEG differences between heavy smoker and nonsmoker subjects. Neuropsychologia, 1968, 6, 381-388.

Brown, B. B. EEG differences between smokers and nonsmokers. In Borgatto, E. G., & Evens, R. R. (Eds.), Smoking, health, and behavior. Chicago: Aldine, 1974.

Bryon, W. S. Hypnosis and smoking. Journal of the American Institute of Hypnosis, 1964, 5, 17-37.

Budzynski, T. H. Biofeedback procedures in the clinic. Seminars in psychology. Grune and Stratton, Inc., 1973, 5, 537-547.

Budzynski, T. H., Stoyva, J., Adler, C., & Mulloney, D. EMG biofeedback and tension headache: A controlled outcome study. Biofeedback and Self-Control, 1973, 253-264.

Claiborn, W. L., Lewis, P., & Humble, S. Stimulus satiation and smoking: A revisit. Journal of Clinical Psychology, 1972, 28, 416-419.

Crasilneck, H. B., & Holt, J. A. Clinical hypnosis application in smoking and obesity problems. Dallas Medical Journal, 1976, 62 (6), 296-302.

Cruickshank, A. The anti-smoking clinic. Lancet, 1963, 2, 353-354.

Danaher, B. G., Lichtenstein, E., & Sullivan, J. M. Comparative effects of rapid and normal smoking on heart rate and carboxyhemoglobin. Journal of Consulting and Clinical Psychology, in press.

Daniel, R. S. Electroencephalographic pattern quantification and the arousal continuum. Psychophysiology, 1965, 2, 146-160.

Dawley, H., Ellithorpe, D., & Tretola, R. Aversive smoking: Carboxyhemoglobin levels before and after rapid smoking. Journal of Behavior Therapy and Experimental Psychiatry, 1976, 7, 13-15.

Domino, E. F. Electroencephalographic and behavioral arousal effects of small doses of nicotine: A neuropsychopharmacological study. Annals of the New York Academy of Science, 1967, 142, 216-224.

Ejurp, B. A proposed medical regimen to stop smoking: The follow-up results. Swedish Cancer Society Yearbook, 1963, 3, 468-473.

Ejurp, B. Treatment of tobacco addiction: Experiences in tobacco withdrawal clinics. In Can We Help Them Stop? Chicago: American Cancer Society, Illinois Division, 1964.

Flaxman, F. Smoking cessation: Gradual vs. abrupt quitting. Paper presented at the meeting of the Association for the Advancement of Behavior Therapy, Chicago, November 1974.

Frank, G. M., Fried, R., & Ashem, B. An improved apparatus for the aversive conditioning of cigarette smokers. Behavior Research and Therapy, 1966, 4, 301-308.

Frankenhauser, M., Myrstem, A. L., Waszak, M., Neri, I., & Post, B. Dosage and time effects of cigarette smoking. Psychopharmacologia, 1968, 13, 311-319.

Gale, A., Dunkin, N., & Coles, M. Variation in visual input and the occipital EEG. Psychonomic Science, 1969, 14, 262-263.

Gallup. Opinion Index Report 3108, June 1974, 20-21.

Garg, M., & Holland, H. C. Consolidation and maze learning: A further study of post-trial injections of a stimulant drug (nicotine). Neuropharmacology, 1968, 7, 55-59.

Geller, I., & Hartman, R. Tranquilizing action of nicotine: Similarity of effects with meprobamate, a minor tranquilizer. Proceedings of the 77th Annual Convention of the American Psychological Association, Washington D. C., 1969, 867-868.

Goldstein, L., & Nelsen, J. M. Behavioral and electrophysiological effects of the "chronic nicotine state" in rats. In National Clearinghouse for Smoking and Health, Directory of on-going research in smoking and health. Arlington, VA.: United States Public Health Service, 1974.

Green, E. Biofeedback for mind-body self-regulation: Healing and creativity. Biofeedback and Self-Control, 1972, 152-166.

Guilford, J. Factors related to successful abstinence from smoking: Final report. Los Angeles: American Institute for Research, 1966.

Hale, G. H. Effects of nicotine and tobacco smoke on the electrical activity of the cerebral cortex and olfactory bulb. British Journal of Pharmacology, 1970, 38, 271-286.

Hardt, J. V., & Kamiya, J. Conflicting results in EEG alpha feedback studies: Why amplitude integration should replace percent time. Biofeedback and Self-Regulation, 1976, 1, 63-75.

Hauser, H., Schwarz, B. E., Roth, G., & Bickford, R. G. Electro-encephalographic changes related to smoking. Electroencephalograph Clinical Neurophysiology, 1958, 10, 576.

Havelick, R. J. The use of biofeedback and twilight learning in the treatment of migraine headache, valium dependency and cigarette smoking. Biofeedback and Self-Regulation. 1977, 2, 297-298.

Hersen, M., & Barlow, D. H. Single case experimental designs: Strategies for studying behavior change. New York: Pergammon Press, 1976.

Hoffstaedt, E. G. Anti-smoking campaign. Medical Officer, 1964, 111, 59-60.

Hunt, W. A., & Bespeloc, D. A. An evaluation of current methods of modifying smoking behavior. Journal of Clinical Psychology, 1974, 431, 438.

Hunt, W. A., & Matarazzo, J. D. Three years later: Recent develop-ments in the experimental modification of smoking behavior. Journal of Abnormal Psychology, 1973, 81, 107-114.

Itil, T. M., Ulett, G. A., Hsu, W., Klingenberg, N., & Ulett, J. A. The effects of smoking withdrawal on quantitatively analyzed EEG. Clinical Electroencephalograph, 1971, 2, 44.

Janis, I. L., & Mann, L. Effectiveness of emotional role-playing in modifying smoking habits and attitudes. Journal of Experimental Research in Personality, 1965, 1, 84-90.

Jarvik, M. E. Further observations on nicotine as the reinforcing agent in smoking. In W. L. Dunn, Jr. (Ed.), Smoking behavior: Motives and incentives. Washington, D. C.: L. Winston & Sons, 1973.

Jarvik, M. E., Glich, S. O., & Nokomura, R. K. Inhibition of cigarette smoking by orally administered nicotine. Clinical Pharmacology and Therapeutics, 1970, 11, 574-576.

Johnson, L., Lubin, A., Naitoh, P., Nute, G., & Austin, M. Spectral analysis of the EEG of dominant and nondominant alpha subjects during waking and sleeping. Electroencephalography and Clinical Neurophysiology, 1969, 26, 361-370.

Johnston, E., & Donoghue, J. R. Hypnosis and smoking: A review of the literature. The American Journal of Clinical Hypnosis, 1971, 13, 265-272.

Kamiya, J. Conditioned discrimination of the EEG alpha rhythm in humans. Abstract of a paper presented at the Western Psychological Association, 1962.

Kamiya, J. A fourth dimension of consciousness. Experimental Medicine and Surgery, 1969a, 27, 13-18.

Kamiya, J. Operant control of the EEG alpha rhythm and some of its reported effects on consciousness. In C. T. Tarts (Eds.), Altered States of Consciousness, New York: John Wiley and Sons, 1969b, 480-501.

Kawamura, H., & Domino, E. F. Differential actions of m and n cholinergic agonists on the brainstem activating systems. International Journal of Neuropharmacology, 1969, 8, 105-115.

Kentzer, C. S. Behavior modification of smoking: The experimental investigation of diverse techniques. Behavior Research and Therapy, 1968, 6, 137-157.

Kentzer, C. S., Lichtenstein, E., & Mees, H. L. Modification of smoking behavior: A review. Psychological Bulletin, 1968, 70, 520-533.

Knapp, D. E., & Domino, E. F. Action of nicotine on the ascending reticular activating system. International Journal of Neuropharmacology, 1962, 1, 333-351.

Knott, J. V., & Venables, P. H. EEG alpha correlates of non-smokers, smokers, smoking, and smoking deprivation. Journal of Applied Behavioral Analysis, 1977, 14, 150-156.

Kothare, V. Application of Yoga, autohypnosis and biofeedback training in the modification of smoking and over-eating behaviors - a pilot study. Procedures of the Biofeedback Research Society Sixth Annual Meeting, Monterey, 1975, 25 (ABST).

Kroger, W. S. Clinical and experimental hypnosis. Springfield: Charles C. Thomas, 1963.

Lamb, W., Ulett, G., Masters, W., & Robinson, D. Premenstrual tension: EEG, hormonal and psychiatric evaluation. American Journal of Psychiatry, 1953, 109, 840-848.

Lambiase, M., & Serra, C. Fumo e sistema mervoso. 1. Modification: dell attivita elettvica corticale da fumo. Acta Neurologica, 1957, 12, 475-493.

Larson, P. S., Haag, H. B., & Silvette, H. Tobacco, experimental and clinical studies a comprehensive account of the world literature. Baltimore: Williams & Wilkins, 1961.

Lawrence, J. Alpha brain waves. New York: Avon, 1972.

Lawson, D. M., & May, R. B. Three procedures for the extinction of smoking behavior. Psychological Record, 1970, 20, 151-157.

Lawton, M. P. Group methods in smoking withdrawal. Archives of Environmental Health, 1967, 14, 258-265.

Leventhal, H. Experimental studies of anti-smoking communications. In E. G. Borgatto & R. R. Evans (Eds.), Smoking, health, and behavior. Chiago: Aldine, 1968.

Lichtenstein, E., Harris, D. E., Brichler, G. R., Wahl, J. M., & Schmahl, D. P. Comparison of rapid smoking, warm smoky air, and attention placebo in the modification of smoking behavior. Journal of Consulting and Clinical Psychology, 1973, 40, 92-98.

Lichtenstein, E., & Kentzer, C. S. Modification of smoking behavior: A later look. In R. D. Rubin, H. Fensterheim, A. A. Lazarus, & C. M. Franks (Eds.), Advances in behavior therapy, New York: Academic Press, 1971.

Lichtenstein, E., Kentzer, C. S., & Himes, K. H. Emotional role-playing and changes in smoking attitudes and behavior. Psychological Reports, 1969, 25, 379-387.

Lichtenstein, E., Kentzer, C. S., & Mees, H. L. Modification of smoking behavior: A review. Psychological Bulletin, 1968, 70, 520-533.

Lilly, J., & Shurley, J. T. Experiments in solitude in maximum achievable physical isolation with water suspension of intact healthy persons. In B. E. Caherty (Ed.), Psychophysiological aspects of space flight. New York: Columbia University, 1961.

Lindsley, D. B. Attention, consciousness, sleep, and wakefulness. Handbook of Physiology, Neurophysiology (Washington, D. C.: American Physiology Society, 1960, 3.

Lubar, J. L., & Bahler, W. W. Behavioral management of epileptic seizures following EEG biofeedback training of the sensorimotor rhythm. Biofeedback and Self-Regulation. 1976, 1 (1), 77-104.

Mann, L., & Janis, I. L. A follow-up study on the long-term effects of emotional role playing. Journal of Personality and Social Psychology, 1968, 8, 339-342.

Marrone, R. L., Merksamer, M. A., & Solzberg, P. M. A short duration group treatment of smoking behavior by stimulus saturation. Behavior Research and Therapy, 1970, 8, 347-352.

McFall, R. M. Smoking - cessation research. Journal of Consulting and Clinical Psychology, 1978, 46 (4), 703-712.

McFall, R. M., & Hammer, C. L. Motivation, structure, and self-monitoring: The role of nonspecific factors in smoking reduction. Journal of Consulting and Clinical Psychology, 1971, 37, 80-86.

McFarland, J. W. Physical measures used in breaking the smoking habit. Archives of Physical Medicine and Rehabilitation, 1965, 64, 323-327.

Meyers, A. W., & Craighead, E. Adaptation periods in clinical psychophysiological research: A recommendation. Behavior Therapy, 1978, 9, 391-403.

Morrison, C. F., & Lee, P. N. A comparison of the effects of nicotine and physostigmine on a measure of activity in the rat. Psychopharmacologia (Berl.), 1968, 13, 210-221.

Murphree, H., & Schultz, R. Abstinence effects in smokers. Federation Proceedings. 1968, 27, 220. (Abstract)

Murphree, H. B., Pfeifer, C. C., & Price, L. M. Electroencephalographic changes in man following smoking. Ann. N. Y. Acad. Sci., 1967, 142, 245.

Nolen, J. D. Self-control procedures in the modification of smoking behavior. Journal of Consulting and Clinical Psychology, 1968, 32, 92-93.

O'Keefe, M. T. The anti-smoking commercials: A study of television's impact on behavior. Public Opinion Quarterly, 1971, 35, 242-248.

Perry, C., & Mullur, G. The effects of hypnotic susceptibility in reducing smoking behavior treated by an hypnotic technique. Journal of Clinical Psychology, 1975, 31 (3), 498-525.

Phillips, C. The EEG changes associated with smoking. Psychophysiology, 1971, 8, 64.

Platt, E. S., Krassen, E., & Mausner, B. Individual variation in behavioral change following role playing. Psychological Reports, 1969, 24, 155-170.

Plotkin, W. B. Long term eyes-closed alpha-enhancement training: Effects on alpha amplitudes and on experiential state. Psychophysiology, 1978, 15 (1), 40-52.

Rimm, D. C., & Masters, J. C. Behavior therapy. New York: Academic Press, 1974.

Roberts, A. H. Self-control procedures in modification of smoking behavior: Replication. Psychological Reports, 1969, 24, 675-676.

Ross, C. A. Report on smoking withdrawal. In Can we help them stop? Chicago: American Cancer Society, Illinois Division, 1966.

Roy, J., & Swillinger, E. Application of learning theory principles to eliminate a cigarette smoking habit: A case study. Psychiatric Forum, 1972, 2, 27-31.

Russel, M. A. Effect of electric aversion on cigarette smoking. British Medical Journal, 1971, 1, 82-86.

Rutner, I. T. The modification of smoking behavior through techniques of self-control. Unpublished master's thesis available from Utah State University, Logan, Utah, 1967.

Schechter, M. D., & Cook, P. G. Nicotine--induced weight loss in rats without an effect on appetite. European Journal of Pharmacology, 1976, 38 (1), 63-69.

Schultz, D. P. Sensory restrictions' effects on behavior. New York: Academic Press, 1965.

Schwartz, J. L., & Dubitzky, M. The results of helping people fight cigarettes. California's Health, 1967, 24, 78-83.

Scott, D., Schwartz, M., Farrant, G., & Spiers, C. The importance of methodology in drug studies using EEG. Electroencephalography and Clinical Neurophysiology, 1974, 37, 670-672.

Scott, G. W., Cox, A. G. C., MaClean, K. L., Prince, T. M., & Southwell, N. Buffered lobeline or a smoking deterrant. Lancet, 1962, 1, 54-55.

Shurley, J. T. The hydro-hypodynamic environment. Proceedings of the Third World Congress of Psychiatry, Volume 13. Toronto, Canada: University of Toronto Press, 1963, 232-237.

Silvette, H., Hoff, E. C., Larson, P. S., & Haag. The actions of nicotine on central nervous system functions. Pharmacological Reviews, 1962, 14, 137-173.

Steffy, R. A., Meichenbaum, D., & Best, J. A. Aversive and cognitive factors in the modification of smoking behavior. Behavior Research and Therapy, 1970, 8, 115-125.

Stephens, R. Psychophysiological variables in cigarette smoking and reinforcing effects of nicotine. Addictive Behaviors, 1977, 2, 1-7.

Stoyva, J., & Budzynski, T. B. Applications of muscle and EEG feedback for anxiety, stress disorders and exploration of conscious states. Mental Health Program Reports, 1975, 6, 109-121.

Streltzer, N. E., & Koch, G. V. Influence of emotional role-playing on smoking habits and attitudes. Psychological Reports, 1968, 22, 817-820.

Suedfelt, P. Sensory deprivation used in the reduction of cigarette smoking: Attitude change experiments in an applied context. Journal of Applied Social Psychology, 1973, 3, 30-38.

Suedfelt, P., & Ikard, F. Attitude manipulation in restricted environments: I.V. Psychologically addicted smokers treated in sensory deprivation. British Journal of Addiction, 1973, 68, 170-176.

Surgeon General's Advisory Committee. Smoking and health. Washington, D. C.: United States Public Health Service, 1975.

Toda, N. Regional differences in the response to nicotine in isolated canine arteries. European Journal of Pharmacology, 1976, 35 (1), 151-160.

Tooley, J. T., & Prott, S. An experimental procedure for the extinction of smoking behavior. Psychological Record, 1967, 17, 204-218.

Turin, A., & Nideffer, R. M. Biofeedback training, migraine-like headaches and smoking: A serendipitous finding. Proceedings of the Biofeedback Research Society, Fifth Annual Meeting, Colorado Springs, 1974, 24 (ABST)

Turle, G. C. An investigation into the therapeutic action of hydroxyzine (atarax) in the treatment of nervous disorders and the control of the smoking habit. Journal of Mental Science, 1958, 104, 826-833.

Turner, D. M. Metabolism of small multiple doses of (14-C) nicotine in the cat. British Journal of Pharmacology and Chemotherapy, 1971, 41, 521-529.

Ulett, J. A., & Itil, T. M. Quantitative electroencephalogram in smoking and smoking deprivation. Science, 1969, 164, 969.

United States Public Health Service. Health consequences of smoking. Washington, D. C.: U.S. Government Printing Office, 1971-1975.

Volavka, J., Crown, P., Dornbush, R., Feldstein, S., & Fink, M. EEG, heart rate, and mood change ("high") after cannabis. Psychopharmacologia, 1973, 32, 11-25.

Volavka, J., Matousek, M., & Roubicek, J. Mental arithmetic and eye openings: An EEG frequency analysis and GSR study. Electroencephalography and Clinical Neurophysiology, 1967, 22, 174-176.

Von Dedenroth, T. E. Further help for the "tobaccomaniac." American Journal of Clinical Hypnosis, 1964a, 4, 322-336.

Von Dedenroth, T. E. The use of hypnosis with "tobaccomaniacs." American Journal of Clinical Hypnosis, 1964b, 4, 326-331.

Von Dedenroth, T. E. The use of hypnosis in 1000 cases of "tobaccomaniacs." American Journal of Clinical Hypnosis, 1968, 3, 194-197.

Von Hofstatter, R. Veber gewohnung undtoleranz beim tobakrauchen. Munch. Med. Wschr., 1934, 81, 439-443.

Watkins, H. H. Hypnosis and smoking: A five session approach. International Journal of Clinical and Experimental Hypnosis, 1976, 24 (4), 381-390.

Wechsler, R. Effects of cigarette smoking and intraveneous nicotine on the brain. Federation Proceedings, 1962, 17, 169 (Abstract)

White, M. E. Cigarette smoking. Medical World, 1962, 97, 489-490.

Whitehead, R. N., & Davies, J. M. A study of methylphenidate and diazepam as possible smoking deterrents. Current Therapeutic Research, 1964, 6, 363-367.

Whitman, T. L. Modification of chronic smoking behavior: A comparison of three approaches. Behavior Research and Therapy, 1969, 7, 257-263.

Wilde, G. J. Letter to the editor. Behavior Research and Therapy, 1965, 2, 313.

APPENDICES

Appendix A

Copy of Newspaper Advertisement

("Want to Quit Smoking")

WANT TO QUIT SMOKING?

If you want to quit smoking and are interested in participating in a program designed to aid you in quitting, contact EARL GRIFFITH, Utah State University, at 752-4100, extension 7753, or 752-8462. Not all people interested will be accepted.

Appendix B

Outline of Initial Intake Procedures

OUTLINE OF INITIAL INTAKE PROCEDURES

1. Phone contact, initiated by the subjects, will be received by the senior author. An office appointment will be scheduled at the convenience of the subject.
2. Introductions will consist of the senior author's name, degree being sought, and an indication to the subjects that this study is being conducted to fulfill the requirements of the Ph.D. program in Psychology at Utah State University.
3. A general review of the Biofeedback Lab and the biofeedback equipment will be presented individually to the subjects in the following manner:

> This is the Exceptional Child Center's Biofeedback Lab. All this equipment you see is designed to measure different physiological parameters. I will briefly review each piece of equipment and its function. If you have any questions, feel free to ask me, and I'll try to answer them. However, if I feel the question could bias the results of the study, I will ask you to hold your question until the completion of the study, at which time I will answer your question.
>
> This machine is an electromyograph. It is designed to measure muscle tension. This is a skin temperature unit, which measure just that--your skin temperature. This piece of equipment is called an electroencephalograph, and it is designed to measure the type, frequency, and amplitude of brain waves which you produce. It will also sound an alarm if you fall asleep, so try and stay awake. This piece of equipment is a heart rate monitor, and this large machine is a physiograph, which allows me to record any of the information on paper. Now, allow me to show you my reclining chair in the next room. Go ahead and have a seat, and I will explain how each piece of equipment is attached.
>
> This additional piece of physiological equipment is, as you know, a blood pressure cuff and a stethoscope. I will take your blood pressure before we begin each session and at the completion of each session. This is the EMG headband, which has three electrodes built into it right here. I will be filling these electrode cups with a

conductive cream which is harmless and very similar to a hand lotion. This electrode attachment will be put around your forehead and fastened at the back of your head like this. Next, we have three small sponge electrodes which are attached to the EEG. They will be dipped into a water, salt, and soap solution, and then placed under the EMG headband like this. You will be able to feel it against your scalp, and I will ask you each time I attach them whether or not you can feel them. One electrode will go above your right ear, one above your left ear, and one in the very back. Next, I'll attach this skin temperature thermister to your left hand, and I would like you to keep your left hand resting on the arm of the chair throughout the session. Finally, the last item to be attached is the heart rate monitor. These electrodes will be attached to your chest.

Upon attaching all the equipment, I will ask you if you are comfortable. You may then adjust the reclining chair so you are comfortable. My only request is that you keep the position of the reclining chair in approximately the same position for each session. I will then go into the adjacent room, close the door, dim the lights, and begin the session. At the completion of the session, I will slowly turn up the brightness of the lights, remove the electrodes, and take your blood pressure. Do you have any questions at this point?

Now, before we go any further, let's talk about the commitments you will be expected to meet. You will be required to come to the lab Monday through Friday at whatever time we agree upon. The sessions will last approximately three quarters of an hour. In addition, you will be required to accurately record, on a data sheet attached to your pack of cigarettes, each occurrence of smoking. The only two other requirements are that: (1) you don't smoke one hour before you come into the lab for a session, and (2) that you write a check worth $25 to a charity of your choice, which I will hold until you complete the study. At that time, I will return the check. The $25 deposit will be required to assure the experimenter that you intend to complete the study. If you fail to complete the study after you have signed the consent form, you will forfeit the $25 to the experimenter.

For each statement, circle the number that shows how you feel about it. Do you strongly agree, mildly agree, mildly disagree, or strongly disagree?

Important: Answer every question.

TEST 2	strongly agree	mildly agree	mildly disagree	strongly disagree
A. Cigarette smoking is not nearly as dangerous as many other health hazards.	1	2	3	4
B. I don't smoke enough to get any of the diseases that cigarette smoking is supposed to cause.	1	2	3	4
C. If a person has already smoked for many years, it probably won't do him much good to stop.	1	2	3	4
D. It would be hard for me to give up smoking cigarettes.	1	2	3	4
E. Cigarette smoking is enough of a health hazard for something to be done about it.	4	3	2	1
F. The kind of cigarette I smoke is much less likely than other kinds to give me any of the diseases that smoking is supposed to cause.	1	2	3	4
G. As soon as a person quits smoking cigarettes he begins to recover from much of the damage that smoking has caused.	4	3	2	1
H. It would be hard for me to cut down to half the number of cigarettes I now smoke.	1	2	3	4
I. The whole problem of cigarette smoking and health is a very minor one.	1	2	3	4
J. I haven't smoked long enough to worry about the diseases that cigarette smoking is supposed to cause.	1	2	3	4
K. Quitting smoking helps a person to live longer.	4	3	2	1
L. It would be difficult for me to make any substantial change in my smoking habits.	1	2	3	4

Here are some statements made by people to describe what they get out of smoking cigarettes. How *often* do you feel this way when smoking them? Circle one number for each statement.

Important: Answer every question.

TEST 3	always	frequently	occasionally	seldom	never
A. I smoke cigarettes in order to keep myself from slowing down.	5	4	3	2	1
B. Handling a cigarette is part of the enjoyment of smoking it.	5	4	3	2	1
C. Smoking cigarettes is pleasant and relaxing.	5	4	3	2	1
D. I light up a cigarette when I feel angry about something.	5	4	3	2	1
E. When I have run out of cigarettes I find it almost unbearable until I can get them.	5	4	3	2	1
F. I smoke cigarettes automatically without even being aware of it.	5	4	3	2	1
G. I smoke cigarettes to stimulate me, to perk myself up.	5	4	3	2	1
H. Part of the enjoyment of smoking a cigarette comes from the steps I take to light up.	5	4	3	2	1
I. I find cigarettes pleasurable.	5	4	3	2	1
J. When I feel uncomfortable or upset about something, I light up a cigarette.	5	4	3	2	1
K. I am very much aware of the fact when I am not smoking a cigarette.	5	4	3	2	1
L. I light up a cigarette without realizing I still have one burning in the ashtray.	5	4	3	2	1
M. I smoke cigarettes to give me a "lift."	5	4	3	2	1
N. When I smoke a cigarette, part of the enjoyment is watching the smoke as I exhale it.	5	4	3	2	1
O. I want a cigarette most when I am comfortable and relaxed.	5	4	3	2	1
P. When I feel "blue" or want to take my mind off cares and worries, I smoke cigarettes.	5	4	3	2	1
Q. I get a real gnawing hunger for a cigarette when I haven't smoked for a while.	5	4	3	2	1
R. I've found a cigarette in my mouth and didn't remember putting it there.	5	4	3	2	1

Indicate by circling the appropriate numbers whether you feel the following statements are true or false.

TEST 4
Important: Answer every question.

	true or mostly true	false or mostly false
A. Doctors have decreased or stopped their smoking of cigarettes in the past 10 years.	2	1
B. In recent years there seem to be more rules about where you are allowed to smoke.	2	1
C. Cigarette advertising makes smoking appear attractive to me.	1	2
D. Schools are trying to discourage children from smoking.	2	1
E. Doctors are trying to get their patients to stop smoking.	2	1
F. Someone has recently tried to persuade me to cut down or quit smoking cigarettes.	2	1
G. The constant repetition of cigarette advertising makes it hard for me to quit smoking.	1	2
H. Both Government and private health organizations are actively trying to discourage people from smoking.	2	1
I. A doctor has, at least once, talked to me about my smoking.	2	1
J. It seems as though an increasing number of people object to having someone smoke near them.	2	1
K. Some cigarette commercials on TV make me feel like smoking.	1	2
L. Congressmen and other legislators are showing concern with smoking and health.	2	1

M. The people around you, particularly those who are close to you (e.g., relatives, friends, office associates), may make it easier or more difficult for you to give up smoking by what they say or do. What about these people? Would you say that they make giving up smoking or staying off cigarettes more difficult for you than it would be otherwise? (Circle the number to the left of the statement that best describes your situation.)

3 They make it much more difficult than it would be otherwise.
4 They make it somewhat more difficult than it would be otherwise.
5 They make it somewhat easier than it would be otherwise.
6 They make it much easier than it would be otherwise.

TEST 1

DO YOU WANT TO CHANGE YOUR SMOKING HABITS?

HOW TO SCORE:

1. Enter the numbers you have circled to the Test 1 questions in the spaces below, putting the number you have circled to Question A over line A, to Question B over line B, etc.
2. Total the 3 scores across on each line to get your totals. For example, the sum of your scores over lines A, E, and I gives you your score on *Health*—lines B, F, and J give the score on *Example*, etc.

								Totals
____	+	____	+	____	=	____		Health
____	+	____	+	____	=	____		Example
____	+	____	+	____	=	____		Esthetics
____	+	____	+	____	=	____		Mastery
A		E		I				
B		F		J				
C		G		K				
D		H		L				

Scores can vary from 3 to 12. Any score 9 and above is *high;* any score 6 and below is *low.* Learn from Part 2 what your scores mean.

TEST 2

WHAT DO YOU THINK THE EFFECTS OF SMOKING ARE?

HOW TO SCORE:

1. Enter the numbers you have circled to the Test 2 questions in the spaces below, putting the number you have circled to Question A over line A, to Question B over line B, etc.
2. Total the 3 scores across on each line to get your totals. For example, the sum of your scores over lines A, E, and I gives you your score on *Importance*—lines B, F, and J give the score on *Personal Relevance*, etc.

								Totals
____	+	____	+	____	=	____		Importance
____	+	____	+	____	=	____		Personal Relevance
____	+	____	+	____	=	____		Value of Stopping
____	+	____	+	____	=	____		Capability for Stopping
A		E		I				
B		F		J				
C		G		K				
D		H		L				

Scores can vary from 3 to 12. Any score 9 and above is *high;* any score 6 and below is *low.* Learn from Part 2 what your scores mean.

TEST 3
WHY DO YOU SMOKE?

HOW TO SCORE:

1. Enter the numbers you have circled to the Test 3 questions in the spaces below, putting the number you have circled to Question A over line A, to Question B over line B, etc.
2. Total the 3 scores on each line to get your totals. For example, the sum of your scores over lines A, G, and M gives you your score on *Stimulation*—lines B, H, and N give the score on *Handling*, etc.

							Totals
A	+	G	+	M	=		Stimulation
B	+	H	+	N	=		Handling
C	+	I	+	O	=		Pleasurable Relaxation
D	+	J	+	P	=		Crutch: Tension Reduction
E	+	K	+	Q	=		Craving: Psychological Addiction
F	+	L	+	R	=		Habit

Scores can vary from 3 to 15. Any score 11 and above is *high;* any score 7 and below is *low.* Learn from Part 2 what your scores mean.

TEST 4
DOES THE WORLD AROUND YOU MAKE IT EASIER OR HARDER TO CHANGE YOUR SMOKING HABITS?

HOW TO SCORE:

1. Enter the numbers you have circled on the Test 4 questions in the spaces below, putting the number you have circled to Question A over line A, to Question B over line B, etc.
2. Total the 3 scores across on each line to get your totals. For example, the sum of your scores over lines A, E, and I gives you your score on *Doctors*—lines B, F, and J give the score on *General Climate*, etc.

							Totals
A	+	E	+	I	=		Doctors
B	+	F	+	J	=		General Climate
C	+	G	+	K	=		Advertising Influence
D	+	H	+	L	=		Key Group Influences
				M	=		Interpersonal Influences

Scores can vary from 3 to 6: 6 is *high;* 5, high middle; 4, low middle; 3, *low.* Learn from Part 2 what your scores mean.

Appendix C-1

Individual Smoker's Self-Testing Kit Data

Subject's Scores on the Smoker's Self-Testing Kit

Test Question	Subtest	Subject's Raw Scores					
		S_1	S_2	S_3	S_4	S_5	S_6
1. Do you want to change your smoking habits?	Health	12*	10*	11*	12	12*	11*
	Example	9	6	11*	7	7	6
	Esthetics	10	10*	11*	9	8	10
	Mastery	12*	10*	11*	12*	11	10
2. What do you think the effects of smoking are?	Importance	11*	8	16*	12*	11*	11*
	Value of Stopping	11*	10*	16*	11	10	9
	Personal Relevance	11*	9	16*	10	11*	9
	Capability for Stopping	5	5	4	6	5	10
3. Does the world around you make it easier or harder to change your smoking habits?	Doctors	6*	5	6	6*	5	5
	General Climate	6*	6*	6*	6*	6*	6*
	Advertising Influence	6*	5	5	5	6*	5
	Key Group Influence	5	4	5	6*	6*	6*
	Interpersonal Influence	4	3	5	5	5	4
4. Why do you smoke?	Stimulation	3	6	11	12*	3	6
	Handling	11	9	8	3	6	7
	Pleasurable Relaxation	13	10	13	10	12*	11*
	Crutch: Tension Reduction	16*	15*	15*	14*	10	9
	Craving: Psychological Addiction	12	13*	15*	10	16*	11*
	Habit	7	11	10	3	5	5

*High score

For Test Question 1 - Scores can vary from 3 to 12. Any score 9 and above is high; any score 6 and below is low. Learn from Part 2 what your scores mean.

For Test Question 2 - Scores can vary from 3 to 12. Any score 9 and above is high; any score 6 and below is low. Learn from Part 2 what your scores mean.

For Test Question 3 - Scores can vary from 3 to 6: 6 is high; 5, high middle; 4, low middle; 3, low. Learn from Part 2 what your scores mean.

For Test Question 4 - Scores can vary from 3 to 15. Any score 11 and above is high; any score 7 and below is low. Learn from Part 2 what your scores mean.

Appendix D

General Background Questionnaire

QUESTIONS FOR THE INTAKE

1. How old were you when you started smoking cigarettes regularly?

   _____yrs. of age.

2. Did you ever try to quit smoking before? (If never, skip to question 5)

   Never_____ Once_____ Twice_____Three or more_____

3. What is the longest period of time you quit smoking completely?

   _____Less than 24 hours

   _____One to six days

   _____One week or more, but less than one month

   _____One month or more, but less than three months

   _____Three months or more, but less than six months

   _____Six months or more, but less than one year

   _____One year or more

4. Have you ever used any particular method or technique to try to quit smoking?

   _____None

   _____Voluntary program (Five-Day Plan, American Cancer Society, etc.)

   _____Commercial program (Smoke Watchers, Schick, etc.)

   _____Drugstore remedy (Nicoban, Bantron, etc.)

   _____Other (Describe)_____

5. On the average, how much do you now smoke per day?

   _____cigarettes per day

   _____cigars/cigarillos per day

   _____pipefuls per day

6. Your sex: _____Male _____Female

7. Your age at last birthday: _____years

8. What is the highest educational level you have completed:

_____Less than high school

_____Some high school

_____High school graduate

_____Some college or specialized school above high school

_____College graduate

_____Some postgraduate work

_____Graduate degree (M.A., M.S., M.S.W., PH.D., M.D., D.D.S., L.L.D., ETC.)

9. What is your occupation?_____

10. Please mention any health problems or current chronic conditions:

_____

SUGGESTED QUESTIONS FOR THE FOLLOW-UP:

1. During and/or immediately after completing treatment, did you quit smoking completely for as long as one week or more?

   _____Yes

   _____No

2. Since completing treatment, how much have you smoked?

   _____I now smoke regularly

   _____I did smoke regularly for some period, but I do not smoke now

   _____I have smoked occasionally

   _____I have not smoked at all

3. If you have smoked since the stop smoking course, please indicate how long a time passed since you quit smoking completely until you first started smoking regularly (at least averaging one cigarette per day.)

   _____months _____weeks _____days

4. On the average, how much do you now smoke per day? If you do not smoke, please write 0.

   _____cigarettes per day
   number

   _____cigars/cigarillos per day
   number

   _____pipefuls per day
   number

5. Would you recommend other smokers to attend the _____ program as conducted by _____

6. Please write below any comments you feel may be helpful to us in our evaluation of the program.

   _____

   _____

   _____

   _____

Appendix E

Consent Form

CONSENT FORM

I hereby consent to participate as an experimental subject in a study of occipital alpha increase training. I understand that I am required to be present at the Biofeedback Lab five (5) days a week for three quarter of an hour sessions for approximately 30 days, and record on the hour my smoking frequency for three months. I am also expected to mail or hand in daily postcards to the experimenter which will indicate the number of cigarettes I smoked that day. The postcards will be provided by the experimenter. Upon the completion of the three month period, I also agree to once a week record on the hour the number of cigarettes I smoked and mail it to the experimenter. I understand this once a week smoking recording will last for 12 weeks. I understand that failure to meet this agreement with more than two excused absences will result in a loss of part or all of my $25 deposit.

I have been informed that my name and identifying information will remain anonymous in any written, oral, or taped communication of the research. I have further been informed that there is no danger of accidental electrical shock, nor any negative side effects as a result of my participation.

Date: _____ Signed: _____

Date Witnessed: _____ Witness: _____

Appendix F

Smoking Frequency Data Sheet

APPENDIX F

SMOKING FREQUENCY DATA SHEET

Name: Date: SIDE 1
6:00- 7:00am
7:00- 8:00am
8:00- 9:00am
9:00-10:00am
10:00-11:00am
11:00-12:00pm
12:00- 1:00pm
1:00- 2:00pm
2:00- 3:00pm
3:00- 4:00pm
4:00- 5:00pm
5:00- 6:00pm
6:00- 7:00pm

SIDE 2
7:00- 8:00pm
8:00- 9:00pm
9:00-10:00pm
10:00-11:00pm
11:00-12:00am
12:00- 1:00am
1:00- 2:00am
2:00- 3:00am
3:00- 4:00am
4:00- 5:00am
5:00- 6:00am
TOTAL:

Appendix G

Average Blood Pressure Data

Average Blood Pressure

Subject	A			B			A			C			D		
	Pre	Post	Degree Change	Pre	Post	Degree Change	Pre	Post	Degree Change	Pre	Post	Degree Change	Pre	Post	Degree Change
S_1	141.88 94.5	137.25 94.0	-4.63 -.5	138.7 96.33	134.0 91.33	-4.7 -5.0	141.3 94	133.3 91	-8.0 -3.0	144 93.7	136.7 98.3	-7.3 +4.6	143.3 98.7	133.7 90.67	-9.6 -8.03
S_2	117.3 80.20	114.8 79.7	-2.5 -.5	123.66 83.0	114.67 79.0	-8.99 -4.00	116.67 78.3	117.33 73	+.66 -5.3	115.33 71.7	113.3 72	-2.03 -.3	116.33 76.67	115.33 78.0	-1.0 +1.33
S_3	129.0 75.75	122.3 70.5	-6.7 -5.25	125.33 71.33	119.33 68.67	-6.0 -2.66	122.5 79.0	117.5 76	-5.0 -3.0	126 79	127.33 75.67	-1.33 -3.33	129.67 82.67	118.67 76.0	-11.0 -6.67
S_4	145.3 82.3	138.7 81.7	-6.6 -.6	141 77.3	136 75.5	-5.0 -1.8	137 76.7	132.7 75.3	-4.3 -1.4	142.7 83.3	140.7 82.33	-2.0 -.97	143.3 88.67	138.3 87.33	-5.0 -1.34
S_5	113.8 71.83	110.3 70.5	-3.5 -1.33	119.6 75.6	114.2 72.8	-5.4 -2.8	114.3 75.6	113.67 67.67	-.63 -7.93	114.7 69.67	112 67.33	-2.7 -2.34	115.67 74	111.3 76	-4.37 +2.0
S_6	133.3 83	123.8 77.8	-9.5 -5.2	129.2 78	127.8 81.6	-1.4 +3.6	122.75 72.3	115.8 72.8	-6.95 +.5	126 71.6	118 70.3	-8.0 -1.3	128.7 79.3	115.0 71.7	-13.7 -7.6

Appendices H Through O Legend

In the following Appendixes (H-O) the numbers shown represent the means and standard deviations of the minute segments: 0-5, 6-10, 11-30 per session per physiological parameters. For the first three phases (Baseline A_1, Smoking B, and Baseline A_2) all sessions are represented on the tables. For the remaining two phases (Feedback C and Fadeout D) only the data for the last three sessions of each phase are indicated on the tables. The small numbers located under each of these groups of means and standard deviations represent the sessions number. Only the last three session's data for the Feedback C and Fadeout D phases is presented on the table due to the author's inability to place all of the data on the available Table size. Appendix O does however indicate the means and standard deviations of the percent time of alpha production (8-12 Hz) per subject for each session of the Feedback C Phase.

Appendix H

Means and Standard Deviations of the Percent Time Alpha Production (8-12 Hz) Per Subject for Each Session

Sub		A_1				B				A_2				C				D											
S_1																													
1-5	mean	92.40	90.70	81.90	92.40	92.00	92.10	89.30		92.00	91.20	93.05		79.10	83.10	92.70		86.00	96.40	91.70									
	SD	3.24	5.00	19.72	2.76	3.14	3.18	8.03		3.90	3.68	3.74		9.88	7.92	4.74		10.04	1.58	4.83									
6-10	mean	95.20	91.10	89.20	91.00	82.20	81.80	79.20		92.50	94.00	93.34		78.30	85.60	92.10		81.40	96.30	92.40									
	SD	2.20	3.73	5.99	2.62	5.53	6.46	3.46		4.14	2.67	3.41		11.23	6.90	7.13		15.84	1.95	9.90									
11-30	mean	94.20	92.53	93.73	91.04	90.53	89.98	91.26		93.63	89.00	91.36		85.10	81.50	93.93		85.70	97.33	93.33									
	SD	2.79	3.23	3.72	4.19	5.80	6.35	5.59		5.19	6.77	6.42		12.41	13.06	7.00		10.24	1.91	14.86									
													Sessions:	(19)	(20)	(21)		(27)	(28)	(29)									
S_2																													
1-5	mean	55.00	93.00	87.10	95.00	84.70	74.70	86.70	94.10	83.60	93.20	91.00		91.20	89.10	93.70		93.20	93.70	95.10		86.50	93.80	87.90					
	SD	6.52	3.40	9.85	6.93	9.17	10.80	8.17	4.04	10.32	4.27	6.07		4.64	7.08	2.79		6.75	4.22	3.14		6.47	5.43	8.09					
6-10	mean	84.50	92.40	93.70	94.50	87.10	78.00	91.60	88.90	73.80	77.60	75.40		86.00	84.60	84.00		97.60	93.80	92.70		89.90	97.10	93.80					
	SD	1.90	4.01	2.45	1.96	6.64	9.65	4.87	4.86	10.81	11.66	7.20		7.15	9.01	11.81		1.43	4.98	6.07		4.16	3.00	4.69					
11-30	mean	83.63	93.13	88.80	83.75	85.73	77.36	80.03	84.05	85.08	82.03	82.90		87.67	87.25	86.10		96.63	98.18	93.99		91.28	96.48	89.20					
	SD	7.95	9.62	5.23	6.44	8.60	11.44	8.41	6.43	8.33	8.67	7.07		5.04	8.05	5.19		4.18	6.97	7.44		5.91	2.26	6.21					
													Sessions:	(30)	(31)	(32)		(38)	(39)	(40)									
S_3																													
1-5	mean	77.00	83.00	88.30	82.00	72.70	85.80	86.00	91.80	89.00	91.60	92.31		63.70	93.80	79.60		89.30	81.10	91.50		78.50	91.30	84.00		93.00	94.00	91.00	
	SD	12.78	14.32	10.11	17.02	7.53	8.68	12.65	7.27	6.97	8.33	7.02		19.14	3.43	10.62		12.46	14.48	7.50		5.58	9.24	15.18		6.19	5.71	9.61	
6-10	mean	76.50	85.50	79.70	65.00	79.70	86.10	85.00	86.50	84.30	92.40	82.31		70.50	70.00	67.20		89.00	58.40	90.70		80.50	94.30	84.60		97.40	93.70	95.20	
	SD	14.34	11.97	29.02	20.56	16.16	10.41	13.28	9.37	4.99	4.84	14.43		9.80	9.82	13.86		10.96	21.62	5.44		5.39	5.42	12.87		2.76	5.48	10.07	
11-30	mean	72.68	75.33	69.00	62.33	76.10	78.03	73.05	78.50	72.10	68.10	72.51		77.35	88.93	82.35		78.50	59.00	82.00		80.70	94.10	87.20		93.45	98.98	90.13	
	SD	12.82	22.27	17.87	19.76	12.64	19.05	13.15	14.80	16.12	20.78	13.86		10.12	9.38	11.49		16.03	22.11	16.51		6.03	7.03	12.29		8.03	1.98	4.43	
																					Sessions:	(23)	(24)	(25)		(31)	(32)	(33)	
S_4																													
1-5	mean	82.50	89.50	83.40		78.10	82.30	85.40	85.10	81.50	83.60	89.80		95.00	80.80	59.10		66.00	54.10	55.70									
	SD	8.78	5.03	8.99		5.02	7.92	4.67	6.37	5.79	8.15	5.01		5.52	11.14	8.77		11.92	19.84	21.03									
6-10	mean	83.80	91.70	79.20		71.20	75.00	86.30	71.50	80.90	84.00	85.60		94.40	76.20	75.56		83.80	74.70	43.90									
	SD	5.09	5.86	12.37		11.04	10.44	5.60	7.89	9.71	9.38	8.95		4.35	20.13	13.90		6.76	16.50	6.62									
11-30	mean	80.68	86.67	85.23		78.40	83.40	84.60	82.85	79.62	84.50	83.85		90.30	87.62	74.30		73.12	64.38	51.98									
	SD	8.35	7.28	6.83		7.87	7.26	14.07	6.02	10.17	5.90	8.65		10.42	9.29	12.13		10.74	15.31	16.41									
													Sessions:	(28)	(29)	(30)		(36)	(37)	(38)									
S_5																													
1-5	mean	80.70	81.80	42.20		78.80	78.10	69.00		78.80	33.00	77.20	79.40	72.90		65.20	74.30	61.90		72.60	75.80	73.70		68.00	72.50	54.00			
	SD	8.33	5.20	11.62		6.05	5.15	7.48		5.55	6.63	8.79	9.58	8.65		16.23	6.85	10.05		5.79	7.36	6.56		10.71	12.09	12.97			
6-10	mean	78.90	80.80	36.40		79.20	75.30	74.30		68.00	42.30	70.70	83.50	57.40		63.80	74.90	61.70		71.20	74.50	77.60		66.50	79.60	76.40			
	SD	8.19	9.73	5.60		4.94	11.21	11.18		7.85	8.64	8.77	9.34	7.43		5.29	9.51	10.22		9.05	11.40	9.45		7.95	9.78	13.43			
11-30	mean	71.13	73.20	54.78		73.13	71.70	70.91		78.95	50.45	80.13	88.18	74.50		63.70	73.88	63.70		77.30	76.25	82.30		64.63	79.65	72.33			
	SD	12.52	10.18	9.36		8.36	8.54	10.89		7.11	9.88	8.39	7.04	8.78		13.37	10.19	9.67		10.97	8.61	7.62		10.15	6.25	10.23			
																			Sessions:	(26)	(27)	(28)		(30)	(31)	(32)			
S_6																													
1-5	mean							61.70	73.70	72.30	76.20		34.50	62.50	54.00	76.20	61.90		67.73	70.10	78.00	58.10		73.90	75.20	77.70		79.40	63.60	63.30	
	SD							11.33	9.45	8.79	6.66		15.62	18.50	7.47	8.05	7.87		12.05	16.25	8.18	14.00		8.80	12.20	15.60		11.64	12.42	17.99	
6-10	mean							66.80	85.20	75.80	75.70		27.60	57.10	42.90	61.80	45.20		66.80	55.00	75.70	57.20		76.30	76.70	79.90		63.60	55.00	79.50	
	SD							8.82	4.94	8.78	3.80		11.09	4.51	13.70	15.31	13.60		13.55	18.20	12.40	7.56		7.96	9.60	12.58		12.42	13.07	14.57	
11-30	mean							67.03	81.68	72.75	69.65		62.68	64.50	45.40	74.00	68.25		70.10	61.85	76.63	69.35		75.30	73.23	81.00		55.00	56.33	74.13	
	SD							6.24	8.05	8.54	13.08		18.42	12.39	17.07	12.27	17.39		13.04	15.07	9.83	14.47		10.19	17.93	11.20		13.07	16.83	10.50	
																					Sessions:	(34)		(37)	(38)	(42)	(47)	(48)	
		1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18										

Appendix I

Means and Standard Deviations of the Amplitude of Alpha (8-12 Hz) Per Subject for Each Session

S3

min		A1				B			A2			C			D		
1-5	mean	25.80	25.00	20.70	27.30	24.40	29.20	26.40	34.80	29.60	32.20	42.40	41.90	31.30	38.50	48.90	44.30
	SD	4.78	4.99	3.68	3.65	4.85	4.54	2.41	3.12	3.48	3.56	9.22	9.06	2.26	7.78	9.31	7.23
6-10	mean	27.30	28.30	23.30	29.10	20.70	21.80	23.50	35.40	31.30	33.35	43.30	40.50	35.10	36.70	50.00	49.80
	SD	5.72	5.36	3.71	5.00	2.63	3.19	2.37	5.91	4.45	3.89	7.65	4.41	6.56	4.22	8.54	8.22
11-30	mean	27.23	28.68	25.38	28.33	23.73	26.95	27.20	39.73	34.73	37.23	54.38	40.83	36.38	40.03	44.00	47.05
	SD	4.63	4.05	4.19	5.12	4.10	4.10	4.22	10.30	3.86	3.54	60.81	9.41	6.34	11.40	6.60	6.90
	Sessions:											(23)	(24)	(25)	(31)	(32)	(33)

S2

min		A1				B				A2			C			D					
1-5	mean	28.80	30.20	31.70	28.80	31.50	34.10	24.90	32.50	30.80	29.70	25.60	34.30	26.30	41.50	38.80	45.40	32.70	37.60	04.34	44.90
	SD	7.41	3.19	8.68	2.90	2.59	4.98	3.87	6.07	6.88	4.35	6.31	4.76	5.98	4.79	4.89	4.40	3.27	6.22	10.46	4.70
6-10	mean	30.10	33.20	30.40	28.20	28.00	31.00	22.40	35.60	29.60	34.00	24.40	38.10	27.00	37.50	40.40	43.70	28.80	37.60	38.90	47.80
	SD	4.28	3.39	6.80	3.85	4.52	6.67	4.67	13.74	6.95	9.01	4.99	9.56	5.75	4.93	5.72	5.50	10.08	6.29	3.11	7.47
11-30	mean	71.55	29.13	33.90	28.69	27.95	33.21	19.88	32.53	26.90	28.30	28.10	37.40	29.13	39.75	45.60	40.78	39.38	40.50	43.68	47.03
	SD	24.07	4.06	5.66	4.36	4.28	13.19	3.81	9.30	4.02	9.41	8.62	7.74	5.62	6.84	5.62	5.62	7.99	7.00	4.46	7.97
	Sessions:															(31)	(32)	(33)	(40)	(41)	(42)

S1

min		A1				B				A2			C			D								
1-5	mean	26.90	37.20	30.80	36.20	29.40	31.90	33.30	30.80	29.00	43.40	32.89	32.50	43.30	35.70	50.10	49.50	38.80	32.90	41.10	40.00	41.20	30.60	36.50
	SD	4.33	6.89	9.58	9.13	9.00	10.02	7.85	3.43	11.79	6.96	4.88	10.69	4.30	5.77	8.61	4.65	13.89	1.29	7.45	17.61	7.71	3.34	6.31
6-10	mean	29.00	36.50	32.00	31.60	34.70	38.30	35.30	29.10	27.20	45.20	33.96	39.10	29.30	46.80	36.70	39.80	44.00	35.10	46.30	35.60	48.40	35.50	34.50
	SD	6.29	6.54	6.00	7.32	11.06	7.23	12.04	3.28	6.84	6.61	6.32	9.02	5.70	19.27	10.17	11.63	4.59	1.73	5.81	3.98	5.48	1.08	2.42
11-30	mean	28.95	33.48	28.35	30.92	31.95	37.33	26.45	34.00	33.78	41.95	32.72	33.83	44.93	37.95	33.50	41.90	48.45	34.78	44.45	43.50	48.73	37.18	38.38
	SD	9.38	12.79	10.80	9.63	8.82	9.95	8.90	8.07	9.72	13.14	8.54	14.35	9.90	8.77	12.27	18.87	10.97	1.28	9.11	13.70	9.70	4.37	5.71
	Sessions:																		(24)	(25)	(26)	(33)	(34)	(35)

S4

min		A1			B				A2			C			D		
1-5	mean	17.30	22.00	23.20	24.00	23.10	23.90	23.60	25.10	24.80	29.00	32.30	39.00	37.70	43.70	38.10	36.00
	SD	3.40	1.88	4.26	1.05	4.82	4.93	1.78	5.51	27.10	10.23	2.41	6.91	4.24	6.70	21.22	6.00
6-10	mean	14.70	21.20	35.80	27.50	25.00	22.50	28.80	25.50	23.50	29.70	36.00	42.20	33.70	37.70	34.50	51.50
	SD	1.05	1.93	18.44	5.40	4.85	3.27	9.46	8.95	3.78	5.45	2.45	2.01	4.92	4.27	8.28	7.70
11-30	mean	16.68	20.63	27.45	25.08	24.68	25.68	23.08	28.76	25.20	27.38	34.50	36.85	42.57	42.70	40.53	44.95
	SD	3.59	3.41	4.36	4.41	6.05	6.92	3.88	8.65	4.15	9.43	9.01	6.60	9.43	7.75	9.57	96.97
	Sessions:											(28)	(29)	(30)	(36)	(37)	(38)

S5

min		A1						B					A2			C			D		
1-5	mean	16.70	13.30	29.40	21.60	19.10	26.80	17.20	39.30	17.50	16.60	15.40	22.90	23.50	21.10	17.50	22.70	17.20	29.00	20.30	23.10
	SD	8.63	1.57	5.79	10.42	2.42	11.36	3.55	5.17	2.99	7.96	2.84	10.10	14.46	3.21	1.35	1.42	3.29	11.49	3.49	5.40
6-10	mean	15.00	12.80	24.50	18.80	18.60	20.80	21.50	39.60	24.90	24.40	39.20	20.40	16.60	21.00	23.10	23.30	16.40	26.50	18.50	20.40
	SD	3.53	1.32	5.10	2.89	1.89	2.62	7.62	3.37	10.42	21.43	13.20	4.12	2.41	2.36	5.99	4.95	2.79	6.55	1.65	4.79
11-30	mean	19.93	14.95	30.75	19.70	21.50	23.34	16.48	30.03	15.03	14.23	15.90	20.80	18.13	21.05	20.00	23.45	17.83	32.23	18.58	27.78
	SD	7.00	4.39	12.35	3.88	4.38	8.19	7.97	5.28	3.32	3.98	5.23	4.33	6.14	4.54	5.80	7.48	4.40	10.84	5.51	10.94
	Sessions:															(27)	(28)	(29)	(31)	(32)	(33)

S6

min		A1				B					A2				C			D		
1-5	mean	23.00	18.20	16.90	24.00	21.20	28.20	22.40	17.00	29.70	34.80	28.50	23.50	23.80	31.70	25.50	31.10	21.40	33.60	43.10
	SD	2.31	2.78	3.84	6.00	4.60	10.20	8.83	2.54	11.41	8.88	6.99	2.51	3.05	12.73	9.69	6.31	4.43	3.78	11.27
6-10	mean	21.60	19.10	20.50	21.30	26.50	19.70	23.00	17.00	21.00	35.90	31.70	29.40	27.50	30.20	28.90	33.50	27.20	30.40	32.10
	SD	1.96	1.73	4.09	4.27	4.35	2.79	4.67	2.54	4.44	7.43	11.45	3.81	2.32	4.64	7.85	10.84	5.45	6.98	5.45
11-30	mean	22.18	17.85	21.98	23.20	22.65	25.63	22.63	15.65	28.00	33.20	34.98	32.70	38.53	32.65	26.48	28.10	26.25	31.60	28.15
	SD	6.69	3.27	3.59	7.56	6.40	6.97	5.84	2.78	11.78	8.46	10.09	[illegible]	12.69	11.16	8.00	7.89	5.42	8.95	6.62
	Sessions:														(38)	(39)	(40)	(46)	(47)	(48)

Sessions 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18

Appendix J

Means and Standard Deviations of the Frequency of Alpha (8-12 Hz) Per Subject for Each Session

Min		A_1				B				A_2			C			D											
S_3																											
1-5	mean	10.36	10.34	10.01	10.32	10.55	10.26	9.98		10.19	10.09	10.14	9.70	10.04	10.28	10.41	10.40	10.25									
	SD	.48	.34	.36	.53	.28	.36	.40		.17	.41	.13	.61	.18	.44	.34	.49	.19									
6-10	mean	10.17	10.18	10.28	10.37	10.12	10.12	10.36		10.41	10.25	10.32	9.81	10.03	10.25	10.36	10.52	10.20									
	SD	.47	.19	.18	.53	.15	.15	.30		.31	.47	.28	.40	.09	.38	.28	.36	.08									
11-30	mean	10.04	10.34	10.10	10.07	10.62	10.52	10.62		10.31	10.31	10.30	10.05	10.32	10.33	10.16	10.75	10.38									
	SD	1.52	.25	.20	.31	.41	.35	.41		.25	.30	.27	.49	.23	.29	.21	.26	.26									
												Sessions:	(23)	(24)	(25)	(31)	(32)	(33)									
S_2																											
1-5	mean	10.43	10.15	10.04	10.07	9.88	10.53	9.81	9.75	9.82	10.01	9.95	10.11	10.01	9.93	9.80	9.48	9.62	9.70	9.85	9.96						
	SD	.53	.42	.18	.18	.31	.45	.47	.30	.21	.33	.37	.29	.21	.21	.26	.33	.25	.27	.34	.39						
6-10	mean	10.35	9.93	10.21	10.21	9.62	10.29	9.84	9.76	10.03	9.09	9.74	10.00	10.16	9.91	9.90	9.59	10.11	9.80	10.09	9.89						
	SD	.45	.36	.20	.22	.42	.31	.37	.40	.19	3.13	.30	.30	.28	.21	.23	.45	.19	.23	.13	.26						
11-30	mean	10.23	13.90	9.83	9.81	9.87	10.23	9.96	9.98	9.93	9.76	9.97	9.97	10.07	9.82	9.62	9.36	9.90	9.80	9.87	9.61						
	SD	.49	.54	.27	1.62	.34	.33	.32	.32	.23	1.56	.23	.25	.28	.23	.33	.32	.19	.22	.21	.31						
															Sessions:	(31)	(32)	(33)	(40)	(41)	(42)						
S_1																											
1-5	mean	10.99	10.62	10.57	10.47	10.58	10.47	10.62	10.41	10.94	10.91	10.66	10.93	10.96	10.82	10.50	10.69	10.78	10.75	10.76	9.59	10.70	10.51	10.43			
	SD	.16	.37	.34	.33	.24	.26	.25	.44	.18	.17	.21	.21	.10	.27	.53	.06	.32	.20	.31	.52	.31	.30	.36			
6-10	mean	10.09	10.59	10.81	10.79	10.38	10.37	10.64	10.67	10.71	10.92	10.67	10.78	10.87	10.88	10.45	11.04	11.01	10.88	10.70	10.34	10.61	10.92	10.37			
	SD	.36	.47	.26	.28	.29	.25	.36	.31	.27	.10	.20	.21	.24	.20	.59	.18	.03	.16	.29	.45	.34	.12	.48			
11-30	mean	10.65	10.84	10.74	10.82	10.43	10.61	10.63	10.59	10.67	10.90	10.68	10.80	11.08	11.03	10.67	11.03	10.83	10.87	10.69	10.33	10.54	10.92	10.45			
	SD	.32	.39	.33	.27	.31	.32	.31	.33	.30	.26	.34	.32	.13	.10	.38	.22	.36	.16	.32	.33	.31	.08	.33			
																		Sessions:	(24)	(25)	(26)	(33)	(34)	(35)			
S_4																											
1-5	mean	10.54	10.02	10.12		9.89	10.14	10.29	10.01	9.76	9.76	9.93	9.73	9.70	9.58	9.58	9.83	9.81									
	SD	.39	.24	.25		.06	.39	.32	.23	.35	.29	.18	.26	.34	.23	.29	.31	.59									
6-10	mean	10.39	9.84	9.93		9.79	10.26	10.58	9.80	9.89	9.93	9.68	9.83	9.54	9.63	9.57	9.82	9.39									
	SD	.28	.22	.39		.34	.34	.26	.27	.41	.49	.30	.24	.38	.28	.23	.23	.34									
11-30	mean	10.15	9.98	10.07		9.90	9.91	10.18	9.91	9.83	9.91	9.89	9.75	9.72	9.62	9.59	9.55	9.43									
	SD	.25	.34	.34		.28	.23	.36	.29	.27	.29	.30	.29	.28	.26	.32	.33	.29									
												Sessions:	(28)	(29)	(30)	(36)	(37)	(38)									
S_5																											
1-5	mean	10.18	10.07	9.52	10.09	9.65	9.66	9.98		9.27	10.28	10.22	10.03	10.22	10.20	9.81	10.11	10.08	10.13	10.02	10.03	9.89					
	SD	.18	.33	.20	.41	.41	.31	.11		.29	.37	.33	.08	.57	.32	.19	.32	.14	.14	.14	.15	.07					
6-10	mean	10.12	9.88	9.33	10.36	9.95	9.95	9.80		9.64	10.12	10.28	9.94	10.13	10.16	9.93	9.99	10.20	10.13	9.92	10.13	10.11					
	SD	.15	.16	.19	.26	.29	.20	.57		.29	.16	.29	.08	.31	.17	.11	.11	.28	.19	.39	.18	.32					
11-30	mean	10.06	9.86	9.59	10.07	10.03	9.94	9.84		9.40	10.26	10.17	9.98	10.26	10.05	9.94	9.88	10.13	10.55	9.82	10.40	10.26					
	SD	.22	.35	.31	.18	.21	.31	.35		.26	.29	.33	.23	.53	.21	.34	.26	.22	.40	.28	.32	.31					
																Sessions:	(27)	(28)	(29)	(31)	(32)	(34)					
S_6																											
1-5	mean							11.13	11.13	10.93	10.67	11.04	11.00	11.04	10.56	11.08	10.59	10.70	10.76	11.17	10.48	9.79	10.98	10.69	10.31	9.93	
	SD							.20	.21	.28	.34	.38	.07	.12	.37	.18	.62	.43	.29	.21	.46	.29	.10	.42	.43	.31	
6-10	mean							10.96	10.83	10.62	10.55	10.96	11.03	11.15	10.59	10.88	10.45	10.82	10.76	10.93	10.83	9.99	10.60	10.12	9.88	10.28	
	SD							.07	.33	.34	.39	.51	.14	.21	.33	.38	.37	.38	.40	.19	.42	.49	.45	.33	.46	.44	
11-30	mean							10.91	10.87	10.54	10.59	10.65	10.95	11.04	10.74	10.86	10.76	10.57	10.67	10.56	10.95	10.22	10.27	9.94	10.09	10.21	
	SD							.25	.37	.29	.49	.35	.21	.25	.35	.33	.40	.31	.46	.30	.31	.43	.41	.25	.54	.36	
																					(38)	(39)	(40)	(44)	(47)	(45)	
Sessions		1	2	3	4	5	6	7	8	9	10	11	12		14	15	16	17	18								

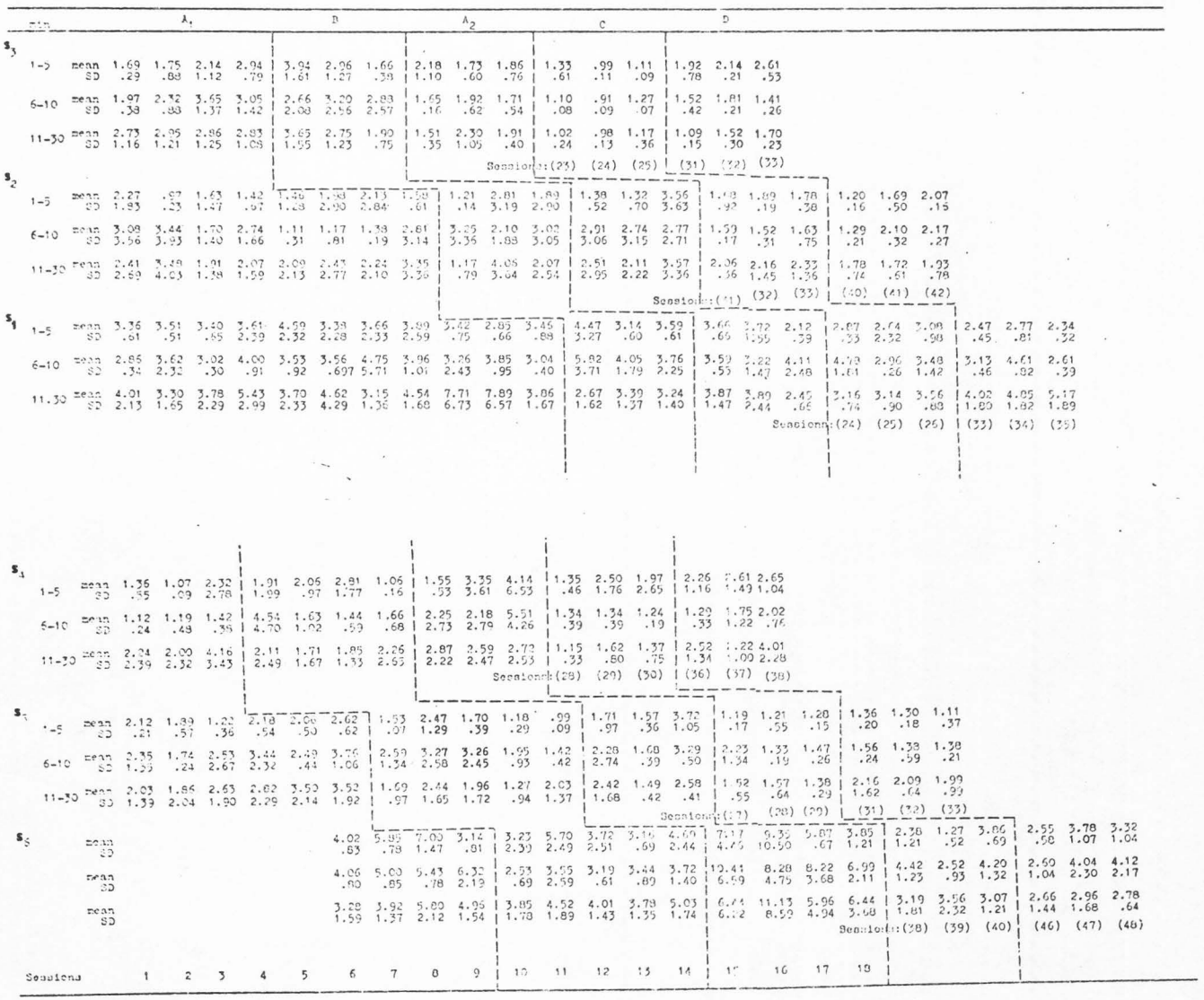
Appendix K

Means and Standard Deviations of the Percent Time Data Production (12-20 Hz) Per Subject for Each Session

	Min		A_1				B				A_2				C				D															
S_3																																			
	1-5	mean	2.50	0.30	1.40	1.40	2.80	2.30	1.40		1.10	1.40	1.23		0.09	1.00	0.70		1.00	0.80	1.60														
		SD	1.43	.95	1.26	1.26	1.87	2.54	2.27		1.85	1.51	1.64		1.52	1.33	1.49		1.15	3.39	1.65														
	6-10	mean	1.10	0.80	1.00	3.30	7.20	3.50	4.30		0.70	0.80	0.75		1.20	0.60	1.10		0.80	1.20	0.20														
		SD	1.45	1.32	1.79	3.68	3.65	1.65	3.59		1.06	1.32	1.20		1.32	.97	1.45		1.40	1.32	0.63														
	11-30	mean	1.08	1.30	0.80	2.64	2.50	1.48	1.00		0.28	1.15	0.72		1.33	0.78	0.60		0.15	0.20	0.20														
		SD	1.59	3.58	1.32	2.91	1.97	1.75	1.97		0.72	1.70	1.20		2.22	2.08	1.41		0.43	0.82	0.56														
														Sessions:	(23)	(24)	(25)		(31)	(32)	(33)														
S_2																																		
	1-5	mean	8.50	2.70	3.60	1.80	0.60	19.40	1.50	1.90	1.00	2.70	2.20		5.40	3.90	1.50		0.70	0.80	0.40		0.20	1.50	0.50									
		SD	3.57	2.36	3.44	1.23	0.97	9.66	2.32	1.60	1.41	3.50	3.05		4.98	3.21	1.35		1.49	1.32	0.84		0.63	1.43	1.08									
	6-10	mean	5.90	1.60	2.40	3.30	1.00	15.90	2.30	2.30	2.00	5.10	2.90		2.30	2.70	3.10		0.60	1.20	2.00		0.80	1.60	0.60									
		SD	6.05	1.43	2.37	2.41	1.15	9.77	1.64	2.31	1.83	4.23	2.20		2.79	2.41	2.32		1.26	1.75	2.36		1.32	1.84	0.00									
	11-30	mean	6.60	10.48	2.85	3.40	1.53	10.13	3.23	4.65	0.78	3.48	3.28		4.38	3.45	4.73		0.40	0.53	0.03		0.28	0.68	0.80									
		SD	5.14	8.52	3.02	3.62	1.74	5.93	3.35	5.21	1.51	2.94	2.98		4.15	4.62	4.24		1.39	1.04	1.42		0.75	1.12	1.34									
																			(31)	(32)	(33)		(40)	(41)	(42)									
																		Sessions:																
S_1																																		
	1-5	mean	15.10	12.20	4.80	18.10	5.30	0.00	5.90	5.00	27.80	4.50	9.86		29.10	4.00	12.60		7.50	13.50	1.00		15.20	5.10	0.30		5.00	1.00	4.70					
		SD	12.31	11.90	5.14	23.10	4.27	0.00	9.67	5.98	10.00	5.25	5.31		17.77	3.43	9.33		8.98	13.04	2.73		4.24	7.43	0.95		8.63	1.66	8.55					
	6-10	mean	14.40	10.60	5.40	20.20	3.90	0.80	7.80	8.80	11.70	5.30	8.87		17.00	20.40	25.80		6.00	30.90	4.00		14.40	2.50	1.40		0.30	0.20	1.70					
		SD	13.08	11.31	4.69	15.50	6.12	1.75	8.56	7.51	5.96	5.71	5.61		7.69	8.00	11.84		6.22	19.99	4.11		5.04	2.22	2.67		0.95	0.42	2.26					
	11-30	mean	15.13	17.48	16.80	20.92	5.95	5.33	20.83	15.70	20.78	29.25	16.41		14.83	6.58	14.63		15.55	32.23	11.63		15.50	1.75	2.30		2.18	0.70	0.63					
		SD	9.80	18.09	11.99	14.68	7.03	6.99	14.02	12.77	16.18	19.87	6.42		9.35	7.45	12.21		13.34	21.48	13.21		5.04	2.01	0.31		3.49	1.44	1.27					
																						Sessions:	(24)	(25)	(26		(33)	(34)	(35)					
S_4																																		
	1-5	mean	5.40	1.40	2.40	0.70	1.70	6.10	2.30		1.10	2.50	1.70		0.00	0.20	0.30		0.40	0.40	6.20														
		SD	3.37	2.55	2.55	1.16	2.26	3.07	2.54		2.02	2.42	1.89		0.00	0.63	0.95		0.84	0.84	12.56														
	6-10	mean	2.85	0.30	2.10	1.70	4.60	10.00	0.60		1.20	2.20	0.50		0.00	0.00	0.20		0.00	0.00	0.00	0.00													
		SD	1.75	0.94	1.79	1.49	2.84	6.81	1.58		2.39	3.00	1.08		0.00	0.00	0.63		0.00	0.00	0.00	0.00													
	11-30	mean	2.96	1.15	3.45	2.08	2.00	4.33	1.08		0.95	1.99	0.50		0.35	0.08	0.05		0.45	0.65	0.13	0.13													
		SD	2.21	1.61	3.32	3.94	2.12	3.03	1.51		2.16	2.18	1.08		0.86	0.47	0.32		1.14	1.12	0.56														
														Sessions:	(28)	(29)	(30)		(36)	(37)	(38)														
S_5																																		
	1-5	mean	9.70	3.80	0.30	0.10	6.40	5.30		3.30	0.50	4.90	12.80	1.70		11.00	2.50	1.60		1.70	1.60	4.40		3.00	5.90	1.40								
		SD	5.38	2.20	0.95	2.73	3.95	8.94		2.79	1.58	2.96	9.58	1.70		11.12	1.90	1.84		1.25	2.84	3.72		2.21	6.94	1.51								
	6-10	mean	7.20	3.00	6.00	8.00	5.70	4.70		4.90	2.20	9.40	7.20	2.10		2.00	3.40	2.10		2.90	3.10	3.70		7.30	6.50	3.30								
		SD	4.10	3.08	14.10	3.54	6.73	5.44		6.10	2.35	5.95	6.58	2.28		2.08	3.31	1.45		4.31	1.97	2.41		11.46	3.54	2.00								
	11-30	mean	5.40	5.10	3.10	5.58	5.40	5.45		2.33	1.20	9.20	4.50	2.73		7.40	4.30	3.93		3.80	2.88	10.60		1.80	8.63	5.25								
		SD	5.41	4.80	3.23	4.75	4.08	8.27		2.22	2.29	8.59	4.13	3.86		13.50	4.08	6.42		3.28	2.59	6.04		2.57	6.19	4.38								
																			(27)	(28)	(29)		(31)	(32)	(33)								
																		Sessions:																
S_6																																		
	1-5	mean						20.70	12.00	13.10	5.40		41.70	16.90	45.00	14.00	33.20		20.20	20.20	23.70	36.40		8.40	2.40	17.00		13.80	11.00	3.30				
		SD						3.03	7.92	7.06	4.17		18.76	19.83	7.78	8.46	16.22		11.52	17.40	10.54	12.03		7.95	3.72	14.92		11.78	8.43	5.08				
	6-10	mean						15.10	6.80	9.00	4.30		47.00	20.20	54.80	31.30	47.10		18.80	36.60	17.40	36.70		16.70	7.90	11.40		3.70	2.50	5.20				
		SD						6.89	4.85	6.43	5.17		16.17	4.32	12.19	14.45	8.86		11.20	27.10	13.25	19.79		6.85	7.75	9.50		3.90	3.47	6.21				
	11-30	mean						16.35	9.05	8.30	8.80		16.33	18.90	57.50	19.28	24.38		20.93	25.80	10.03	16.33		15.58	11.93	6.18		2.35	11.62	5.65				
		SD						6.99	6.99	8.11	9.20		15.85	10.91	10.66	10.88	18.65		12.97	19.41	8.09	14.88		11.29	19.79	6.67		3.36	18.52	6.27				
																							Sessions:	(38)	(39)	(40)		(46)	(47)	(48)				
Columns			1	2	3	4	5	6	7	8	9		10	11	12	13	14																	

Appendix L

Means and Standard Deviations of the Microvolts of Muscle Tension Per Subject for Each Session


A1
B
A2
C
D
S3
S2
S1
S4
S5
Sessions

Appendix M

Means and Standard Deviations of the Heartbeats Per Minute Per Subject for Each Session

		A_1				B				A_2				C				D											
S_3																													
1-5	mean	60.00	58.00	66.50	67.50	67.00	63.50	69.00		63.00	62.00	62.50		80.00	88.00	83.00		91.00	83.00	83.50									
	SD	2.83	0.00	.71	.71	1.41	3.54	1.41		1.41	1.41	1.50		0.00	0.00	5.66		4.24	2.83	2.12									
6-10	mean	57.33	59.00	67.00	67.00	71.33	69.00	73.00		60.67	59.67	60.19		84.67	86.00	76.67		89.33	78.33	80.00									
	SD	.58	.00	2.00	2.00	2.31	4.58	1.41		1.15	1.53	1.31		1.53	2.00	1.53		2.08	1.53	1.73									
11-30	mean	57.50	57.50	65.00	64.00	71.30	69.00	74.00		60.60	57.10	58.87		84.70	80.80	74.90		82.00	76.50	81.40									
	SD	1.08	1.51	1.15	2.23	2.58	1.94	1.73		1.43	1.88	1.13		2.00	3.61	1.91		1.56	2.37	3.63									
													Sessions:	(23)	(24)	(25)		(31)	(32)	(33)									
S_2																													
1-5	mean	91.00	85.50	92.00	80.50	90.00	110.00	86.00	86.00	88.50	84.50	93.00		96.50	96.00	90.50		104.00	102.00	95.50		111.00	96.00	101.00					
	SD	1.41	.71	4.24	.71	.00	0.00	0.00	.00	.71	.71	1.41		3.54	0.00	2.12		0.00	0.00	2.12		1.41	0.00	0.00					
6-10	mean	93.33	87.67	90.67	91.33	91.00	112.67	86.67	90.33	94.00	88.67	98.33		93.67	91.67	93.33		103.00	102.33	97.67		111.33	97.67	98.33					
	SD	.58	.58	.58	1.53	2.52	1.53	2.52	1.53	2.65	3.21	1.53		2.52	2.08	.58		1.00	1.15	.58		.58	.58	.58					
11-30	mean	92.90	87.60	89.30	82.60	91.20	111.70	88.50	85.50	92.30	88.70	95.50		93.00	96.10	91.20		99.00	99.20	95.50		108.50	93.70	98.00					
	SD	1.85	2.12	2.71	1.84	1.06	1.95	3.10	3.46	1.70	2.54	3.31		1.60	2.88	1.32		2.18	2.04	1.84		1.43	2.95	1.41					
																	Sessions:	(31)	(32)	(33)		(40)	(41)	(42)					
S_1																													
1-5	mean	83.00	82.00	91.00	83.00	87.00	89.00	76.00	88.50	81.00	79.00	83.93		93.00	84.00	82.00		84.00	83.50	81.00		88.00	71.00	82.00		81.00	92.00	91.00	
	SD	1.41	0.00	1.41	0.00	0.00	1.41	0.00	2.12	0.00	1.41	1.13		1.41	1.41	0.00		1.41	2.12	1.41		0.00	1.41	1.41		1.41	1.41	0.00	
6-10	mean	88.67	84.67	91.00	80.67	87.00	93.00	77.00	87.33	80.67	78.67	84.77		97.67	83.00	89.67		85.33	84.00	83.33		85.67	71.67	80.33		75.33	90.00	91.33	
	SD	3.79	3.51	10.54	2.31	3.46	3.61	1.00	1.15	1.53	.58	1.33		3.21	2.65	5.86		1.53	1.41	.58		1.53	1.15	1.53		1.53	0.00	1.15	
11-30	mean	75.50	81.70	91.00	80.00	81.60	87.20	77.00	82.30	77.50	78.70	81.63		96.10	90.70	89.00		84.00	84.00	83.70		83.56	70.70	78.10		81.50	88.20	91.00	
	SD	3.78	3.06	3.02	.94	1.35	1.83	1.00	2.00	1.65	2.00	1.03		1.79	1.77	3.87		2.67	2.36	1.77		1.88	2.16	1.73		2.01	1.14	1.07	
																					Sessions:	(24)	(25)	(26)		(33)	(34)	(35)	
S_4																													
1-5	mean	78.00	78.00	74.00		71.50	77.50	75.00	79.00	72.00	79.00	76.50		89.00	87.00	88.50		82.50	89.00	81.50									
	SD	1.41	1.41	1.41		2.12	.71	1.41	1.41	0.00	1.41	.71		1.41	0.00	3.53		.71	1.41	2.12									
6-10	mean	77.00	79.00	74.33		72.67	80.00	76.30	80.67	72.30	78.30	77.67		93.00	88.67	92.33		83.67	88.00	84.00									
	SD	.99	0.00	1.53		.58	.99	1.15	.58	1.53	.58	1.53		.99	.57	.58		1.53	.99	1.73									
11-30	mean	75.10	80.00	72.40		72.60	79.00	74.20	81.00	71.00	77.00	77.20		88.10	86.40	85.40		79.30	82.10	77.60									
	SD	1.52	1.49	1.51		1.71	.04	1.14	1.25	1.33	1.49	1.69		3.73	2.99	3.89		2.41	2.81	1.96									
													Sessions:	(28)	(29)	(30)		(36)	(37)	(38)									
S_5																													
1-5	mean	67.50	76.00	72.50	74.00	62.50	76.00		74.50	76.50	69.00	83.50	79.50		68.50	72.51	76.50		74.00	85.50	83.00		62.50	78.00	77.50					
	SD	2.12	2.83	3.54	1.41	.71	2.83		.71	2.12	1.41	.71	.71		.71	.71	.71		.00	2.12	1.41		.71	1.41	.71					
6-10	mean	66.30	75.60	69.67	72.00	61.33	74.30		85.67	83.30	82.60	95.00	93.67		63.30	70.32	77.33		74.30	82.67	82.67		65.67	79.67	78.00					
	SD	2.08	1.82	.58	.00	.58	1.53		7.09	6.67	8.96	2.64	7.02		2.08	1.44	1.15		2.31	2.00	2.08		2.31	4.16	1.73					
11-30	mean	64.70	73.20	71.60	73.00	63.30	76.70		78.10	88.30	79.60	93.50	8.40		63.90	68.44	73.00		79.00	76.40	83.70		66.20	77.80	79.20					
	SD	1.57	2.57	2.17	0.00	2.70	2.36		3.93	4.92	5.93	5.99	5.64		2.81	1.62	1.15		2.26	2.59	1.06		1.14	1.87	1.87					
																	Sessions:	(27)	(28)	(29)		(31)	(32)	(33)					
S_6																													
1-5	mean						83.00	70.50	63.00	67.00	72.00	66.00	67.00	71.00	69.00	66.00	61.50	62.50	72.50	69.50	67.00	71.50	70.50	54.00	62.21					
	SD						0.00	.71	0.00	.00	.00	1.41	1.41	.00	1.41	.00	.71	.71	.71	3.54	2.83	2.12	.71	.00	.00					
6-10	mean						84.30	68.67	62.67	64.00	74.67	74.30	70.67	73.67	78.33	66.00	64.67	62.30	71.33	72.00	66.00	69.30	68.00	54.30	61.20					
	SD						1.53	2.52	2.08	1.73	2.08	4.50	4.50	1.53	.58	.99	2.52	1.15	1.53	.99	1.73	2.08	1.73	.58	.77					
11-30	mean						81.50	68.60	59.70	65.10	75.30	73.40	70.20	74.70	75.20	64.30	63.00	60.10	71.20	71.90	65.00	64.80	65.00	55.60	60.33					
	SD						1.72	1.82	1.77	2.42	2.45	3.81	2.09	2.06	3.65	1.49	2.36	1.29	2.15	1.97	1.66	1.87	1.49	1.71	1.31					
																				Sessions:	(28)	(30)	(30)		(46)	(47)	(48)					
Sessions		1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18										

Sin		A				B				A_2			C			D										
S_3																										
1-5	mean	88.98	90.62	93.08	93.19	93.00	91.68	94.64		91.50	93.60	92.40	94.18	94.9	92.95	94.30	92.65	91.35								
	SD	.75	.15	.22	.56	.43	.51	.10		.17	.13	.14	.06	.1	.23	.29	.22	.28								
6-10	mean	90.65	90.75	94.26	93.90	94.40	91.04	94.22		91.80	93.82	92.70	94.20	95.14	93.78	94.99	93.40	91.80								
	SD	.18	.13	.19	.11	.18	.18	.29		.00	.06	.02	.00	.10	.18	.36	.27	.11								
11-30	mean	90.93	91.05	94.81	93.62	95.40	90.93	93.93		93.15	93.59	93.13	93.84	95.1	93.95	95.13	93.89	92.32								
	SD	.10	.12	.13	.26	.39	.56	.27		.65	.20	.40	.16	.1	.16	.14	.23	.24								
												Sessions:	(23)	(24)	(25)	(31)	(32)	(33)								
S_2																										
1-5	mean	91.62	90.27	92.48	87.38	93.20	94.62	92.80	93.56	93.16	90.68	94.39	91.94	91.10	92.12	93.24	93.34	94.04	91.60	92.68	93.98					
	SD	.53	.26	.25	.10	.12	.21	.00	.08	.22	.10	.31	.13	.12	.22	.26	.21	.08	.21	.17	.06					
6-10	mean	92.36	90.88	92.80	88.00	93.68	94.52	92.80	93.80	93.28	91.38	94.27	92.18	91.36	92.60	93.74	93.71	94.24	91.94	92.74	94.00					
	SD	.10	.15	.28	.40	.06	.08	.00	.30	.14	.15	.22	.06	.08	.11	.13	.10	.16	.13	.13	.00					
11-30	mean	93.39	91.53	93.09	89.56	93.84	94.73	92.89	91.16	93.35	91.06	95.04	92.32	91.37	92.74	94.05	93.72	94.43	92.59	93.06	93.98					
	SD	.56	.22	.10	.26	.11	.37	.10	.30	.11	.10	.24	.12	.09	.16	.16	.10	.17	.40	.13	.07					
															Sessions:	(31)	(32)	(33)	(30)	(40)	(41)					
S_1																										
1-5	mean	92.39	91.64	92.23	90.98	92.48	90.23	90.41	91.86	91.33	91.66	91.62	91.50	89.60	92.49	93.72	91.76	92.86	94.43	93.87	94.77	93.72	91.23	94.08		
	SD	.45	.25	.10	.59	.46	.35	.35	.44	.31	.46	.30	.45	.91	.45	.21	.45	.16	.16	.21	.28	.23	.50	.15		
6-10	mean	92.84	91.93	92.10	91.90	93.25	91.03	90.66	92.62	91.92	92.42	92.08	91.31	90.81	93.07	94.16	92.66	93.28	94.68	93.96	95.12	94.18	91.72	95.09		
	SD	.21	.11	.23	.14	.08	.22	.09	.11	.10	.15	.14	.28	.18	.13	.08	.13	.17	.10	.10	.10	.11	.12	.13		
11-30	mean	92.25	91.87	93.39	92.40	93.78	92.02	90.25	92.71	91.53	92.32	92.10	91.31	90.78	92.90	94.06	93.14	91.28	94.55	93.66	95.03	93.53	91.10	94.99		
	SD	.27	.25	.20	.19	.58	.26	.28	.11	.36	.15	.80	.55	.20	.18	.23	.15	14.27	.10	.20	.09	.31	.23	.23		
																		Sessions:	(24)	(25)	(26)	(33)	(34)	(35)		
S_4																										
1-5	mean	89.35	92.84	90.13		88.54	90.24	88.87	89.35	91.32	91.03	92.42	90.49	91.78	92.06	92.75	90.16	89.63								
	SD	.18	.37	.08		.37	.34	.67	.46	.37	.29	.50	.45	.30	.16	.08	.13	.17								
6-10	mean	89.76	93.61	90.75		87.44	89.96	88.81	90.31	91.70	91.30	92.95	91.20	91.38	92.00	94.41	91.28	89.40								
	SD	.27	.13	.61		.63	.32	.32	.14	.33	.11	.26	.00	.32	.09	.29	.33	.00								
11-30	mean	90.37	93.80	91.09		88.17	90.40	89.34	91.05	91.41	91.07	93.00	92.06	92.45	92.77	94.72	92.05	90.72								
	SD	.87	.92	.59		.54	.29	.42	.22	.24	.34	.31	.66	.56	.27	.27	.24	.35								
												Sessions:	(28)	(29)	(30)	(36)	(37)	(38)								
S_5																										
1-5	mean	85.92	88.27	84.03		86.84	86.80	91.06		82.43	80.01	79.00	83.00	87.59		79.06	85.56	90.32		87.72	93.95	91.97		83.90	86.32	90.79
	SD	.04	.21	.85		.78	.70	.09		1.05	.75	.00	1.38	.40		.36	.55	.43		1.90	.79	.63		.19	.64	.53
6-10	mean	96.16	89.39	85.76		88.19	87.70	91.08		84.16	83.82	78.90	86.70	87.76		78.96	86.54	91.01		89.50	92.29	92.76		84.59	88.19	91.58
	SD	.22	.44	.61		.26	.11	.23		.18	.10	.00	.19	.37		.16	.13	.13		.42	.23	.09		.18	.29	.06
11-30	mean	86.50	90.36	87.59		89.15	87.34	90.57		84.81	82.51	78.27	86.77	86.12		78.07	86.53	91.10		90.82	92.94	93.21		87.53	90.08	91.36
	SD	.18	.23	.85		.23	.55	1.72		.31	.38	.37	.58	.88		.44	.73	.14		.26	.16	.17		1.45	.49	.15
																		Sessions:	(27)	(28)	(29)		(31)	(32)	(33)
S_6																										
1-5	mean						89.22	93.04	90.85	92.93		91.67	89.24	89.17	91.25	91.30		92.76	93.55	90.79	93.11		95.96	94.54	92.66		93.71	93.88	94.70
	SD						.77	.39	.31	.15		.29	.23	.07	.15	.19		.26	.24	.24	.23		.08	.13	.08		.19	.24	.19
6-10	mean						93.36	93.29	91.06	93.20		91.32	88.94	88.37	91.24	91.40		93.10	93.29	90.77	92.61		96.08	94.58	92.62		93.46	93.32	94.45
	SD						.17	.15	.16	.13		.68	.39	.50	.47	.13		.11	.12	.52	.14		.10	.10	.10		.09	.14	.14
11-30	mean						90.37	93.03	90.46	93.42		92.09	88.90	87.41	89.84	91.32		93.19	93.98	90.96	92.77		96.16	94.66	93.13		93.23	93.48	93.42
	SD						.18	.21	.93	.33		.49	.29	.14	.76	.19		.14	.35	.33	.15		.09	.12	.28		.09	.19	.67
																					Sessions:	(34)	(39)	(40)		(46)	(47)	(48)
Sessions		1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18							

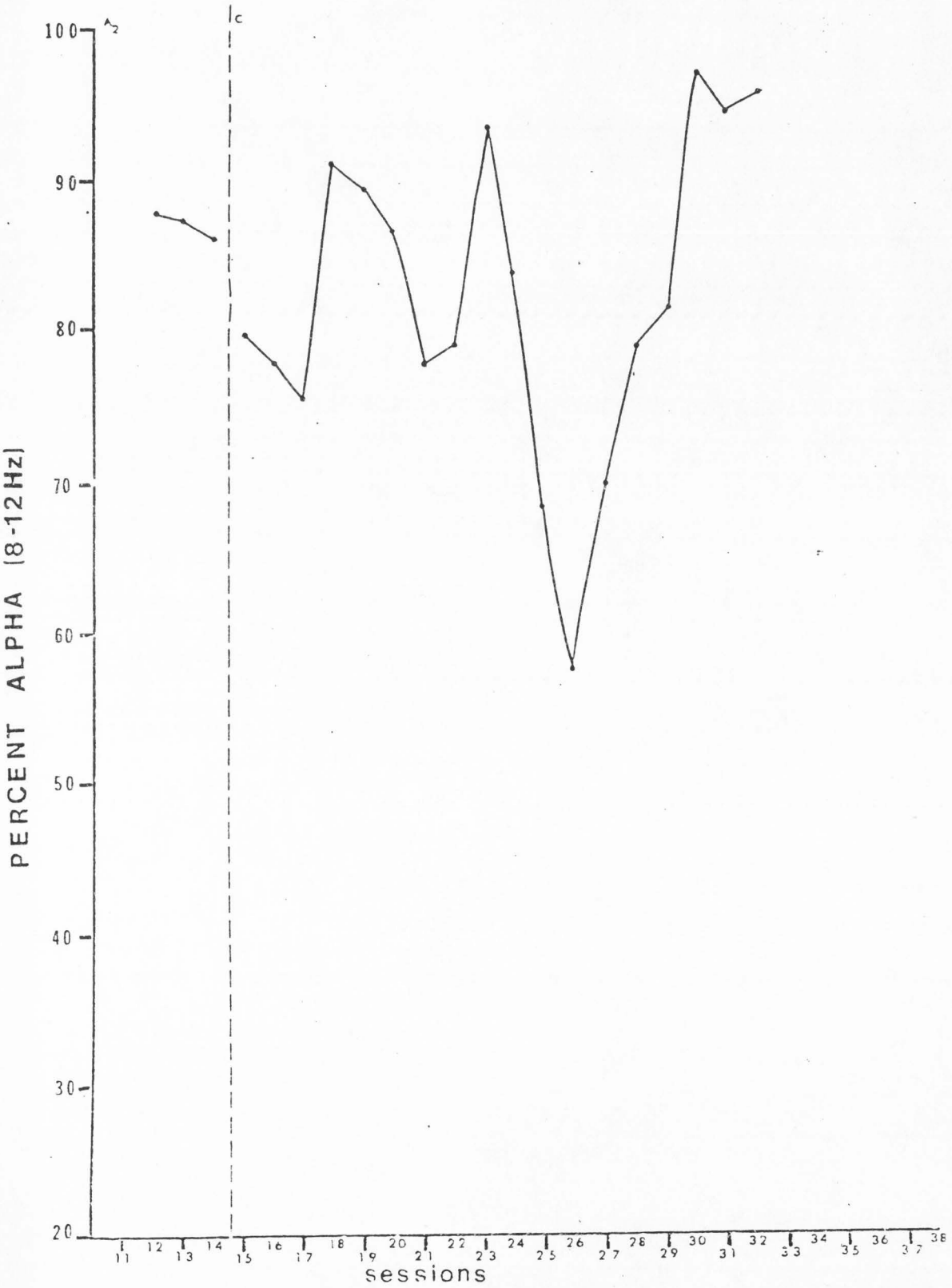
Appendix O

Means and Standard Deviations of the Percent Time of Alpha Production (8-12 Hz) per Subject for Each Session During the Feedback C Phase

Bin																														
S_3																														
1-5	mean	83.60	85.70	88.30	72.10	79.60	85.00	51.00	89.10	79.10	83.10	92.70																		
	SD	9.13	6.07	7.13	8.41	8.50	7.44	12.00	5.09	9.88	7.92	4.74																		
6-10	mean	88.10	91.00	89.70	80.50	72.50	87.00	59.80	84.40	78.30	85.60	92.10																		
	SD	4.53	5.15	5.68	3.38	6.20	6.51	10.11	9.44	11.23	6.90	7.13																		
11-30	mean	90.40	85.55	79.45	75.58	80.60	93.25	64.00	87.70	85.10	85.50	93.93																		
	SD	5.64	3.31	15.69	7.54	6.15	5.23	13.00	11.82	12.40	13.06	7.00																		
S_2																														
1-5	mean					89.30	85.80	97.40	88.60	78.70	83.10	78.70	81.10	88.50	91.30	75.60	51.70	37.80	07.72	88.70	93.30	93.70	95.19							
	SD					4.92	9.07	5.50	4.35	8.49	6.21	5.01	3.98	7.35	4.14	11.64	25.03	29.24	18.80	4.67	6.75	4.22	3.14							
6-10	mean					92.10	74.50	73.80	93.10	85.20	91.60	79.70	82.00	91.10	93.40	64.40	59.40	65.60	82.60	93.10	97.60	93.60	92.70							
	SD					4.63	9.60	12.00	5.26	10.37	3.74	5.08	7.16	3.41	5.38	13.16	11.68	18.15	11.58	4.53	1.43	4.96	6.67							
11-30	mean					79.85	77.05	75.63	90.88	89.13	86.40	77.93	78.95	93.43	84.93	68.35	57.30	69.05	78.43	81.08	96.63	92.18	93.96							
	SD					8.98	6.96	12.59	6.22	5.09	5.72	5.51	9.56	5.73	8.93	15.57	12.46	15.93	15.64	9.55	4.18	6.87	7.44							
S_1																														
1-5	mean								73.40	94.00	94.20	93.00	72.60	78.50	91.30	84.00														
	SD								12.46	5.01	9.71	8.83	7.57	5.58	9.24	19.18														
6-10	mean								81.80	93.50	94.90	91.80	75.00	80.90	94.30	84.80														
	SD								13.05	4.35	4.35	7.87	5.68	5.30	5.42	12.90														
11-30	mean								90.30	94.13	95.70	95.33	78.10	80.70	94.10	85.70														
	SD								7.56	6.86	5.77	6.09	5.66	6.03	7.63	14.94														
S_4																														
1-5	mean	78.20	73.40	77.00	73.90	63.60	60.10	81.00	78.40	75.50	79.70	77.10	64.60	55.70	82.00	61.60	37.00	56.20	95.00	80.80	59.10									
	SD	8.46	7.41	9.20	8.84	15.94	6.37	10.09	5.17	5.02	11.94	8.96	8.28	15.88	8.11	10.38	20.41	18.30	5.52	11.14	8.77									
6-10	mean	81.10	77.40	78.40	87.00	79.30	73.30	90.50	75.50	81.40	86.80	75.70	72.00	74.00	80.60	72.50	69.30	76.60	94.40	76.12	75.56									
	SD	5.76	5.02	12.45	5.01	9.24	10.65	4.48	5.19	5.46	6.71	7.89	11.22	6.69	9.39	12.47	13.25	10.25	4.35	20.13	13.50									
11-30	mean	81.30	83.28	86.55	73.53	82.08	78.68	87.35	76.48	78.13	84.95	75.78	68.60	51.30	78.68	75.76	72.60	74.80	90.30	87.62	79.30									
	SD	3.10	7.99	8.08	15.81	9.55	6.72	7.19	7.07	6.36	6.14	9.28	10.66	17.86	13.77	11.77	16.75	14.29	10.42	9.29	12.13									
S_5																														
1-5	mean					74.00	76.30	73.20	77.30	51.90	51.60	20.40	48.40	53.90	51.00	23.70	72.60	75.80	73.70											
	SD					8.07	6.11	6.30	6.99	10.77	13.64	10.34	17.12	11.87	8.68	9.21	5.79	7.36	6.36											
6-10	mean					78.70	68.50	75.80	78.70	52.20	66.60	33.30	55.70	51.80	55.10	20.10	71.20	74.50	77.60											
	SD					7.97	12.41	8.26	3.43	9.79	16.50	14.39	11.54	10.77	10.05	5.93	9.05	11.40	9.45											
11-30	mean					80.80	70.30	75.70	79.10	59.18	64.63	44.54	51.00	55.45	56.20	29.83	77.30	76.25	82.30											
	SD					8.39	6.32	5.69	6.62	14.27	13.19	14.23	16.66	14.02	14.83	12.20	10.99	8.81	7.62											
S_6																														
1-5	mean									23.00	59.60	59.80	78.40	70.20	74.30	81.00	73.50	76.10	76.80	69.70	71.70	80.80	75.00	74.10	58.00	77.60	73.90	75.20	77.70	
	SD									16.50	17.30	8.39	7.11	7.96	14.38	9.14	11.19	7.92	9.74	6.06	10.59	8.83	13.25	9.45	14.49	6.04	8.86	12.20	15.00	
6-10	mean									60.00	48.00	60.20	83.60	81.00	67.00	79.80	81.10	78.30	76.60	77.00	80.90	91.20	72.50	89.50	81.60	76.50	76.30	76.70	73.[illegible]	
	SD									14.89	15.54	10.58	5.00	4.76	14.24	8.55	8.10	6.58	6.24	5.71	9.90	6.21	6.50	6.48	10.44	10.02	7.56	9.60	13.[illegible]	
11-30	mean									73.38	71.70	66.88	80.75	81.58	73.30	73.50	72.50	77.38	73.38	75.95	75.45	82.43	70.58	73.53	75.90	74.25	75.30	73.[illegible]	[illegible]	
	SD									11.62	12.71	11.14	14.10	14.14	11.23	8.92	8.69	7.51	9.86	7.52	10.51	7.82	11.50	12.79	11.62	13.76	10.18	11.[illegible]	11.[illegible]	

Sections	11	12	13	14	15	16	17	18	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36	37	38

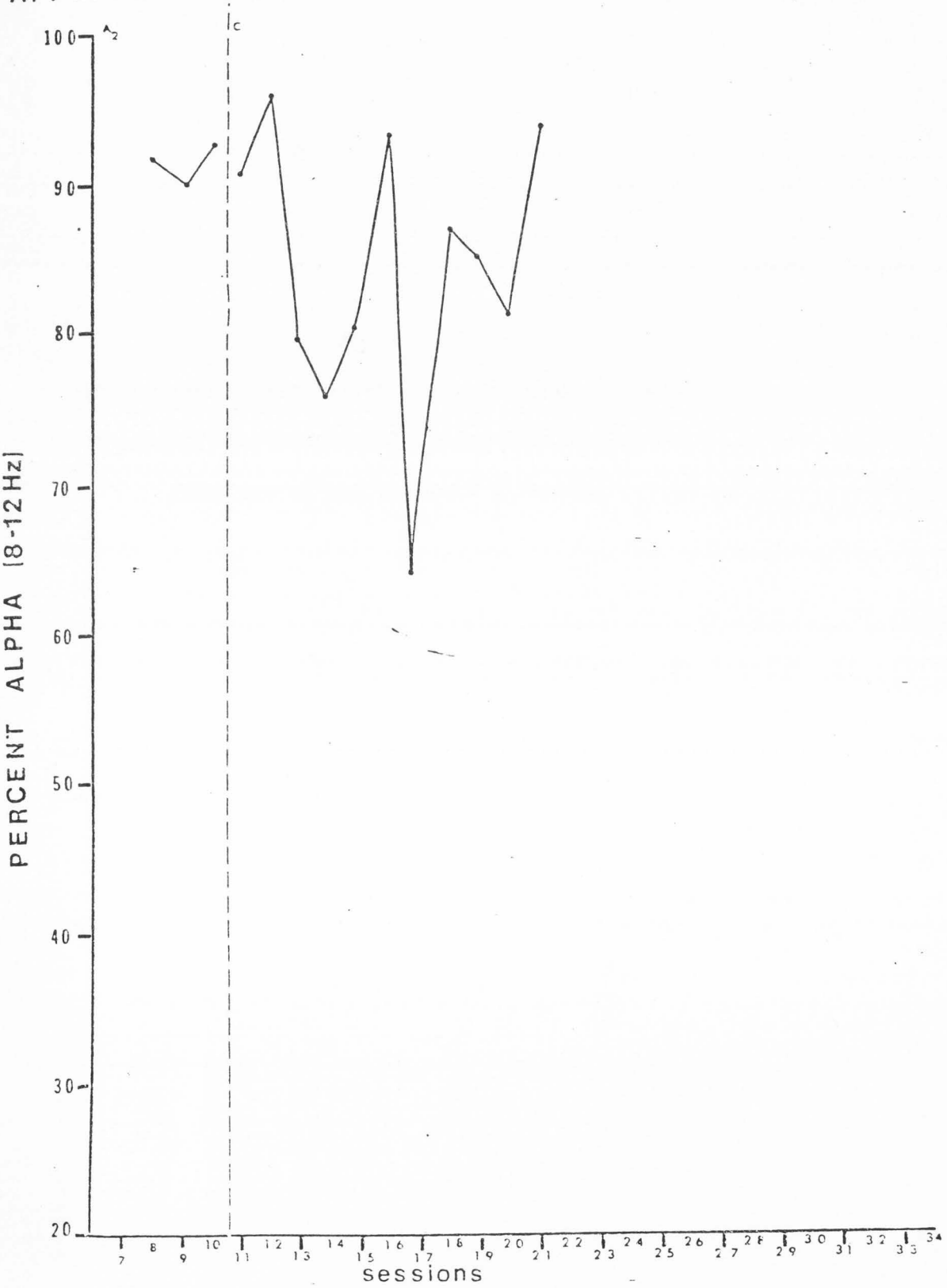
APPENDIX:O-I SUBJECT:2



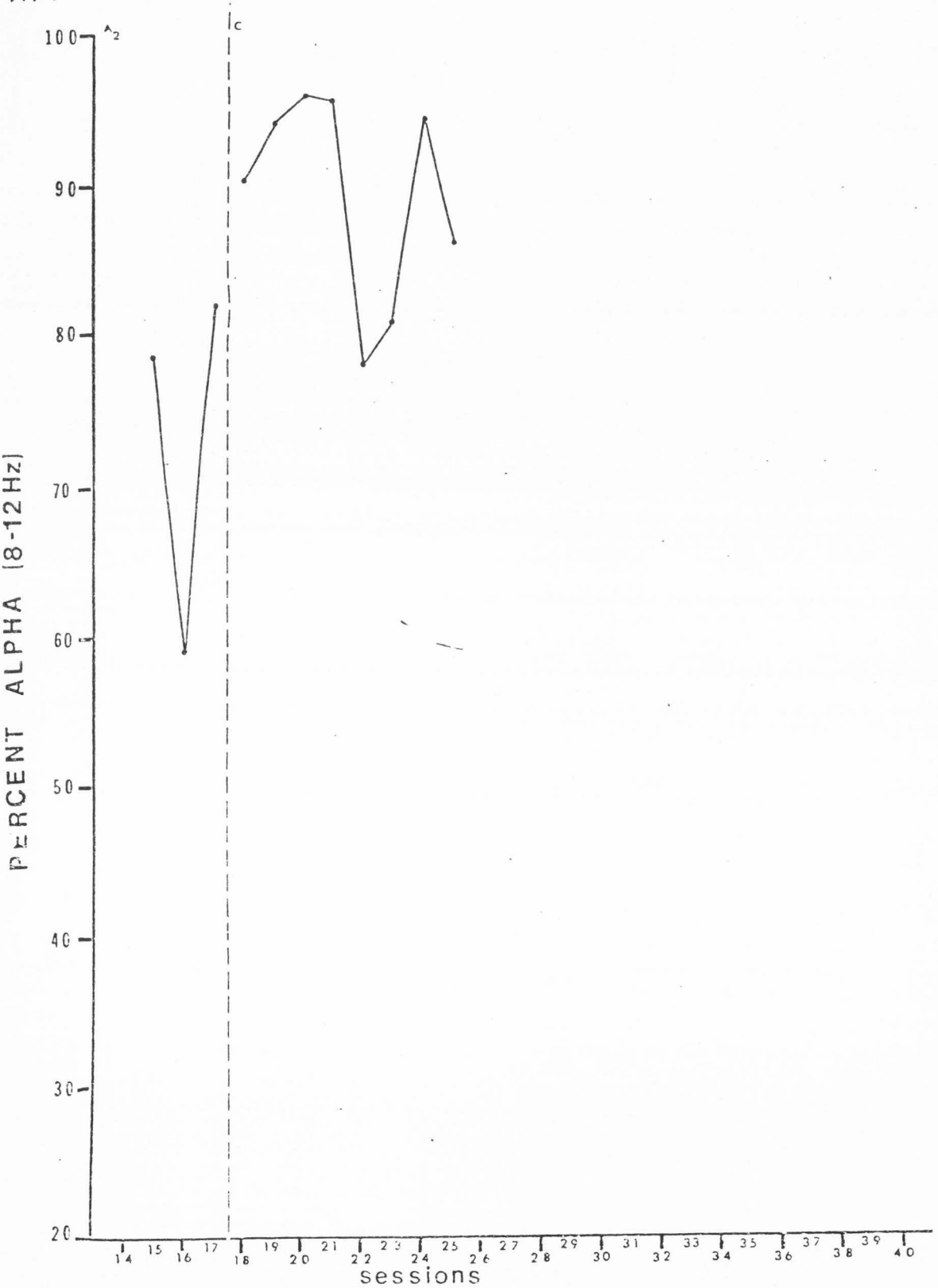
Appendix P

Subject Three's Mean Percent Time of Alpha (8-12 Hz) Production for Minutes 11-30 per Session During Baseline A_2 and Feedback C Phases.

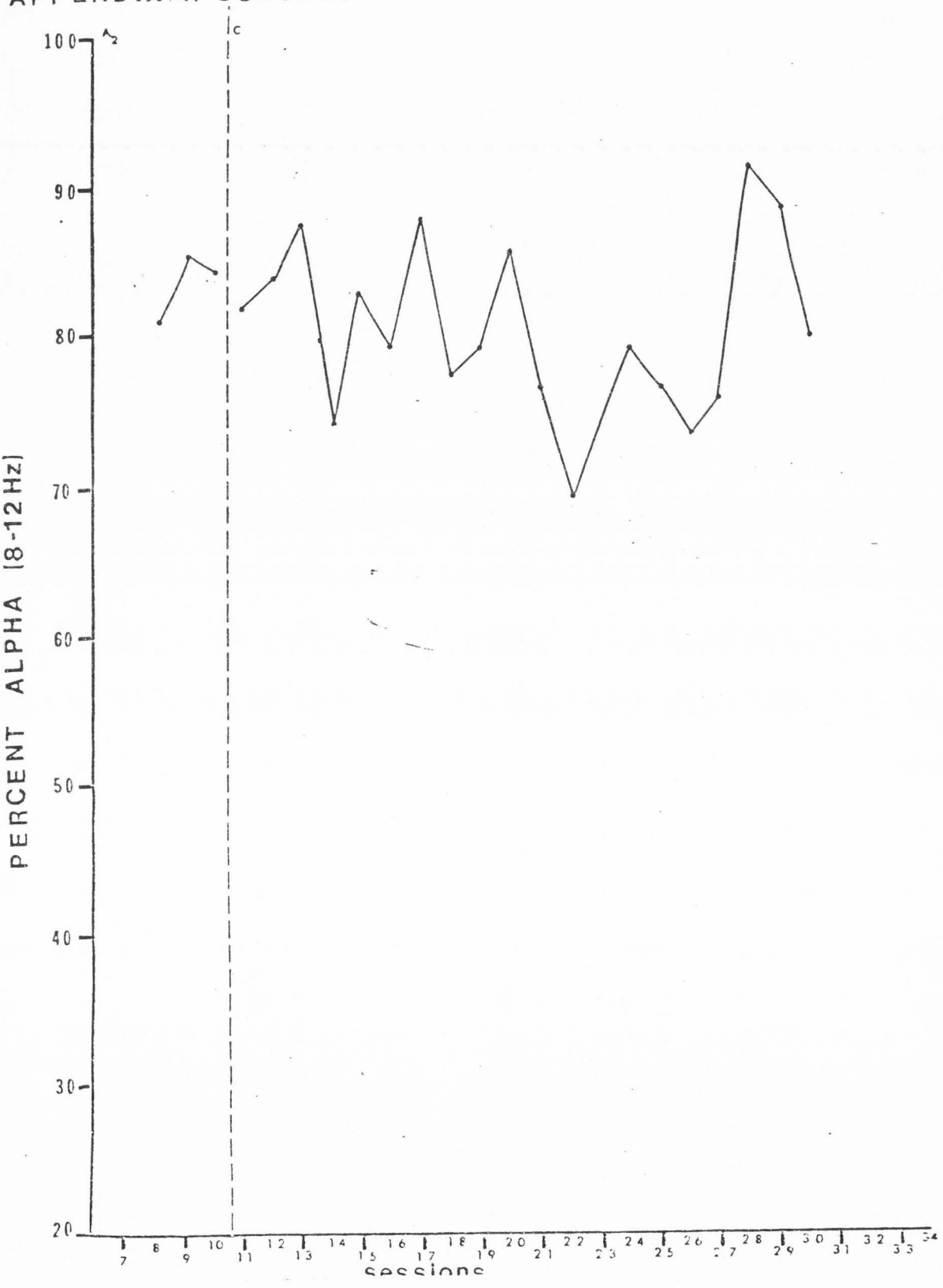
APPENDIX: P SUBJECT:3



APPENDIX :Q SUBJECT: 1



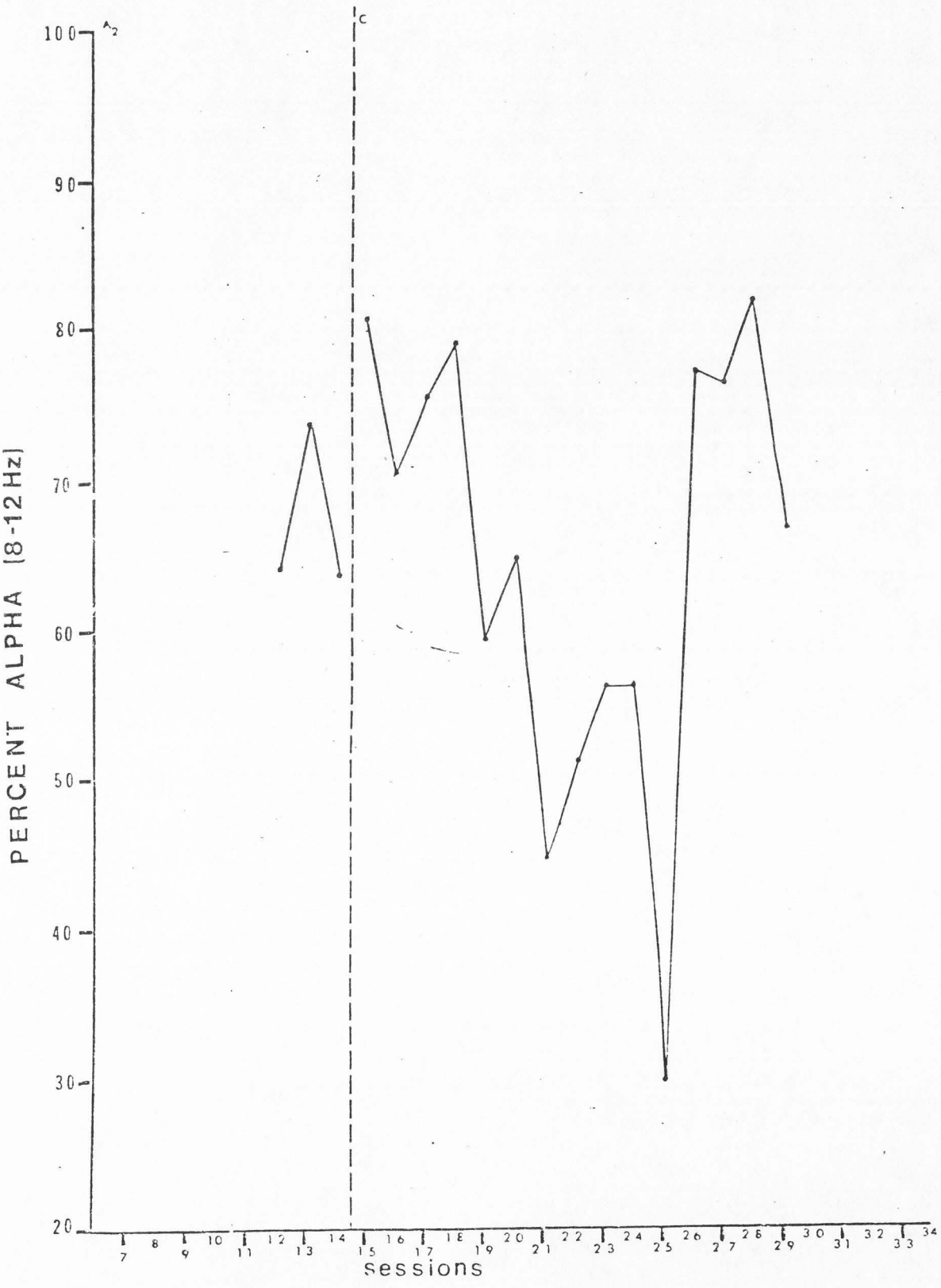
APPENDIX :R SUBJECT:4



Appendix S

Subject Five's Mean Percent Time of Alpha (8-12 Hz) Production for Minutes 11-30 per Session during Baseline A_2 and Feedback C Phases

APPENDIX :S SUBJECT:5



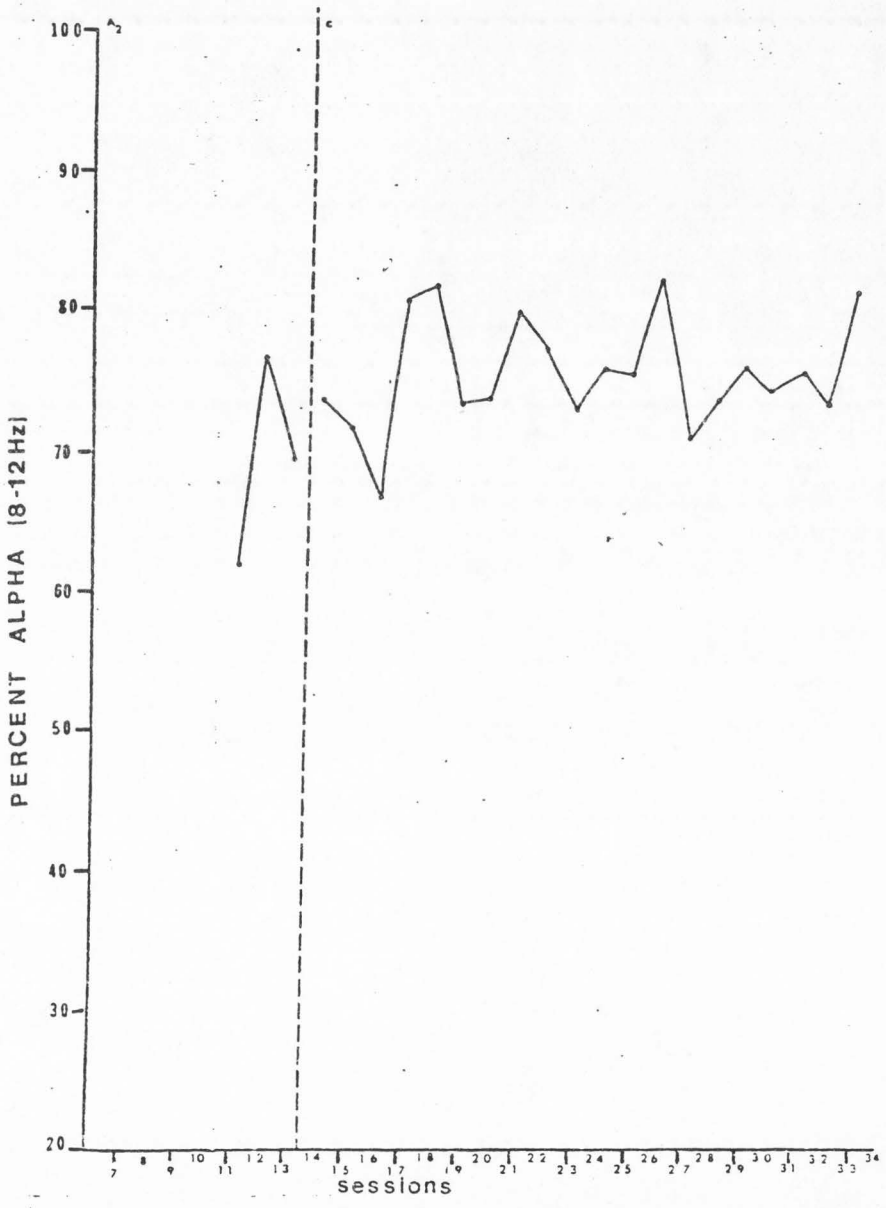
Appendix T

Subject Six's Mean Percent Time of Alpha (8-12 Hz)

Production for Minutes 11-30 per Session During

Baseline A_2 and Feedback C Phases

APPENDIX T SUBJECT: 6



VITA

Earl E. Griffith

VITA

EARL E. GRIFFITH

PERSONAL DATA

Place of Birth: Brownstown, Pennsylvania
Date of Birth: May 21, 1952
Marital Status: Single

Current Business Address: Psychology Department
Utah State University, UMC 28
Logan, Utah 84322

Business Telephone: 801 - 752-4100, extension 7253
Home Phone: 801 - 752-8462 or 717 - 656-6795

EDUCATIONAL BACKGROUND

Ph.D. - Utah State University, Analysis of Behavior
planned completion date: June 1, 1979

Dissertation: "Biofeedback: A Substitute for Smoking."
Data was collected at Utah State University, Exceptional Child Center.

M.A. - Middle Tennessee State University, Clinical Psychology, 1975.

Thesis - "Teacher Modification of Nonverbal Behavior Through the Reinforcement of Related Verbal Behavior: A Single Subject Experiment."

B.S. - Middle Tennessee State University. Major - Psychology, Minor - English, Sociology, 1974.

A.S. - Cumberland Junior College, Lebanson, Tennessee. Major - Biology, 1972.

EMPLOYMENT AND EXPERIENCES

1973-1974 Middle Tennessee State University, Research Assistant to Dr. Jack Schnelle, (Behavior modification with children).

1974-1975 Research Coordinator, Behavior Intervention Team Program, Rutherford County Guidance Center, Murfeesboro, TN. (Behavior modification in the natural environment).

1974-1975 Specialized training, Child Intervention Program, White Ave., Nashville, TN. Under Dr. Linda McClean (Shaping techniques used in working with autistic behavior).

1976-1977 Clinical Practicum Internship at the Exceptional Child Center, Utah State University. Experiences included serving as intake coordinator of multidisciplinary team, diagnostic assessment, educational programming, behavior management programming, parent/child counseling, principal/teacher consulting, assessment and treatment techniques. Under Phyllis Cole and Dr. Sebastian Striefel.

1977-1978 Biofeedback Research Coordinator at the Exceptional Child Center, Utah State University. Experiences included grant writing, consultation of experimental design paradigms, use of EEG, EMG, blood pressure, skin temperature biofeedback training with a variety of clinical problems in both retarded and non-retarded individuals, and supervision of graduate and undergraduate students in using biofeedback training procedures.

June, 1977-July, 1978 Staff Psychologist at the Exceptional Child Center, Utah State University. Experiences including serving as intake supervisor and coordinator of multidisciplinary team, diagnostic assessment, educational programming, behavior management programming, parent/child counseling, principal/teacher consulting, assessment and treatment techniques. Under Dr. Phyllis Cole, and Dr. Sebastian Striefel.

December, 1978-June, 1979 Assistant Coordinator of the Achievement Place Program, Psychology Department, Utah State University. Experiences include the training of Teaching-Parents, conducting workshops, psychological consulting for group homes, providing group and individual therapy to adolescents, and dealing with public relations.

GUEST SPEAKER

Autistic Behavior and a Behavior Modification Approach to Treatment. Presented to Maplewood High School, Maplewood Lane, Nashville, TN. Under request of Mr. Edward Adelman, Principal.

Biofeedback: Its Uses in Gaining Self-Stress Control of Physiological Parameters. Presented to undergraduate psychology students at Utah State University. Under request of Mrs. Helen Tucker, Professor.

PUBLICATIONS

Griffith, Earl E., Schnelle, J., McNees, P., Bissinger, C., and Huff, T. "Elective mutism in a first grader: The remediation of a complex behavior problem." The Journal of Abnormal Child Psychology, 1975, 230-238.

PAPERS PRESENTED

Griffith, E., Kosloski, K., Schnelle, J. The efficacy of a self-recording behavior technique in increasing reading speed and comprehension. Reviewed by Journal of Psychological Reports.

Schnelle, J., Griffith, E., Huff, T., McNees, P., and Thomas, M. Paraprofessionals as behavior change monitors and analysis of community mental health outpatients. Reviewed by Journal of Mental Health Technology.

Griffith, E., and Schnelle, J. Increasing appropriate eating behavior by decreasing self-stimulation in autistic children. Reviewed by Journal of Autism and Childhood Schizophrenia.

Griffith, E. Teacher modification of nonverbal behavior through the reinforcement of related verbal behavior: A single subject experiment. (Accepted for presentation at the Tenth Annual Colloquim in Psychology, May 3, 1975, at Muhlenberg College, Allentown, Pa.

Griffith, E., and Crossman, E. Biofeedback: A substitute for smoking. Presented at Rocky Mountain Psychological Association Convention, Thursday, April 6, 1978, Denver, Colorado.

WORKING PAPERS

Biofeedback: A Substitute for Smoking

E.M.G. Training: A Biofeedback Approach to Treating Hyperactivity

GRANT WRITING

Biofeedback: A Substitute for Smoking. Funded by the Utah Lung Association, November, 1977 to June, 1978.

GRANT SUPPORT (team member of the following grant):

Controlling Hyperactivity in Children Through Relaxation Training. Dr. Sebastian Striefel, Principle Investigator, Exceptional Child Center, Utah State University, 1977-1978.

CONSULTING POSITIONS

1978 Identification of emotionally disturbed, intervention strategies, manual development, in-service for the Preston School District, Preston, Idaho. Responsible to Dr. Blair Henderson.

TEACHING EXPERIENCE

1977 Behavior Modification, Psychology 372
Utah State University
Logan, Utah

1978 Designing Programs to Work with the Emotionally Disturbed, Psy 690
Utah State University
Logan, Utah

REFERENCES

Marvin Fifield, Ph.D.
Director, Exceptional Child Center
Utah State University
Logan, Utah 84322

Glendon Casto, Ph.D.
Associate Director
Exceptional Child Center
Utah State University, UMC 68
Logan, Utah 84322

Edward Crossman, Ph.D.
Associate Professor
Psychology Department
Utah State University
Logan, Utah 84322

Phyllis Cole
Coordinator of Clinical Services
Exceptional Child Center, UMC 68
Utah State University
Logan, Utah 84322

Christa Peterson, Ph.D.
Assistant Professor
Psychology Department
Utah State University
Logan, Utah 84322

Richard Baer, Ph.D.
Exceptional Child Center, UMC 68
Utah State University
Logan, Utah 84322